

ANNALS
OF
OTOLOGY, RHINOLOGY,
AND
LARYNGOLOGY.

VOL. XII.

DECEMBER, 1903.

No. 4.

XLVI.

PARAFFIN PROSTHESIS.

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When in May, 1899, by injecting melted paraffin into the scrotum of an emasculated young man, the masses appeared so like testicles that the applicant was enabled to pass a medical examination as to fitness for military duty, *Gesung Zeit. für Heilk.*, 1900, established a new era in prosthetic surgery.

He suggested that it might be used to raise the cheek after removal of the upper jaw, to fill a cavity in bone after operations for necrosis, to obtain a movable joint after resection to fill the defect in a trephined skull, to support the weight of the blood in varicose veins by making a firm swelling of paraffin around the vein, to cure incontinence of feces or incontinence of urine in the female by injecting paraffin under the prolapsed mucous membrane, to keep back a

hernia, to fill out the flatness of the chest after breast operations.

It has further been used to fill depressions after frontal sinus and mastoid operations.

To raise sunken cicatrices on the face, the result of tooth suppurations in the lower jaw, to make a foundation for an artificial eye, to prevent regeneration of nerves after resection, to raise mucous membrane over shrunken inferior turbinated bodies in ozena.

And above all to improve the appearance of depressed noses.

Dangers in the use of paraffin are due to faulty technic. Thoroughly aseptic precautions must be taken.

Embolism has followed the injection of paraffin into a vein. This first happened to Pfannenstiel, *Centralblatt für Gynak*, 1902, who injected 30 ccm. of paraffin, having a melting point of 115° F. into the loose cellular tissue of a woman suffering from bladder prolapse. She was sent directly to the railway station, and on the way, had a pulmonary embolism with the characteristic symptoms of cough, increased respiration, dyspnea and cyanosis. After suffering for a week the trouble gradually subsided. Hurd and Holden, *Medical Record*, July 11, 1903, report a case of embolism of the central artery of the retina following paraffin injection into the nose. It puzzles one to understand how an embolus could get from a systemic vein into the central artery of the retina without being caught in the capillaries of the pulmonary circulation.

During the injection the patient suddenly began to rub his right eye and complained that he could not see with that eye. A little later ecchymosis appeared about the tip of the nose, indicating that a vein had been punctured. Examination of the eyes 25 minutes after the injection showed the pupil of the right eye large and not responsive to light. The patient had a subjective sense of objects swimming about in the entire field of vision but objectively he was unable to distinguish between light and darkness. The media were clear, the retina not hazy and the retinal veins normal. The main inferior branch of the central artery of the retina and its divisions were empty and collapsed. The main superior

branch contained some blood, but when gentle pressure was made upon the eyeball the blood column here broke up and the blood flowed back into the central artery. Digitalis and amyl nitrite were given at once. Repeated and continued attempts were made by massage to force the embolus forward into one of the branches of the central artery, so as to restore vision to part of the field, but without success. There was no subsequent improvement in vision. Abscess has resulted from injection or hyperinjection.

In the only case that has come under my observation, a brother rhinologist injected paraffin through the mucous membrane to raise the skin of a depressed nose. Suppuration followed and most of the paraffin escaped but sufficient inflammation ensued to leave a fair result.

Undue spreading of paraffin while injecting for improvement of saddle-back nose can be avoided by the assistant's making strong lateral pressure with an index finger placed on either side of the dorsum of the nose as high as the inner canthi, while the operator makes pressure just above the point where he wishes the skin to be raised.

In case of paraffin being misplaced, one must remove it by surgical means. In one such case I tried to soften the mass by extreme heat and then aspirate, but I had to resort to a curette to get it away.

WHAT BECOMES OF THE PARAFFIN?

It is probable that the very hard paraffin, such as that used by Eckstein, which has a melting point of 136° F. becomes encapsuled, while those with a melting point of 104° to 115° F. very slowly become permeated with leucocytes and tissue cells are replaced by fibrous tissue.

When paraffin is being injected the overlying skin becomes much blanched, but reaction soon sets in, and the part becomes reddened by dilatation of the superficial vessels. This redness corresponds to the tension to which the overlying skin is subjected and it may persist from a few hours to many months.

It has been suggested in a condition such as this, that, inasmuch as the x-ray will blanch unsightly red scars by

producing a hyperplasia of the connective tissue cells, which squeezes the blood out of the vessels, that it should cause a speedy disappearance of paraffin erythema.

PREPARATION OF THE PARAFFIN.

The paraffin which I have used has a melting point of 112° F. The ordinary paraffin of commerce has a melting point of about 128° F., and roughly speaking 4 parts of such paraffin to which are added 5 parts of albolene give a mechanical mixture having a melting point of about 112° F. This is placed in a test tube, thoroughly sterilized and laid aside till required.

INJECTION PROCEDURE.

Any steel-barreled, solid-piston hypodermic syringe with a large calibre needle (serum needle) will do. It is preferable, however, to have a coarse screw worm on the piston rod, so that the semi-solid paraffin can be slowly and steadily ejected. The syringe is boiled, the paraffin is melted in a water-bath and the field of operation prepared by scrubbing with green soap, ether and alcohol.

The paraffin is drawn into the syringe till it is about $\frac{3}{4}$ full, the needle is attached and a few drops of hot sterile water are drawn up to fill the needle. This last maneuver prevents a mass of paraffin from plugging the needle by cooling there more quickly than in the loaded barrel. The syringe is then placed in sterile water which is kept at temperature of 120° F. till ready for use.

The assistant then places an index finger on either side of the depressed nostril and makes firm lateral pressure so as to prevent any malplacement of the paraffin.

The needle punctures the skin about $\frac{1}{2}$ of an inch below the lowest point of the depression and is carried upward subcutaneously to its upper limit. The operator then making firm pressure on the dorsum of the nose above and between the assistant's index fingers slowly empties the syringe. As the space fills the needle is gradually withdrawn leaving its deposit of paraffin as it recedes.

The paraffin sets quickly and any necessary moulding should be done at once.

When small quantities are injected there is very little reaction. I am in the habit of using not over 20 to 30 min. at one injection, even if the deformity is uncorrected because it can be repeated each week until the nasal outline is satisfactory.

Except in a small child no anesthetic is required.

Where cicatricial adhesions exist these must be subcutaneously divided and paraffin at once injected to separate the raw surfaces. The needle puncture is sealed with collodion and ice-cloths may be applied for a few hours, but when injecting the smaller quantities the reaction is so slight that it need not interfere with the patient's daily routine.

The two cases illustrated show clearly the possibilities of paraffin prosthesis, both were traumatic, in the one no adhesions existed, in the other much scar-tissue bound the integument to the underlying bone.

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XLVII.

A MODIFICATION OF THE KRIEG OPERATION FOR
DEVIATED SEPTUM.

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The indication for operative interference of the septum narium in most cases is a lack of sufficient patulency of the nasal passages.

Posteriorly, where there is more room laterally, even a considerable deviation or spur will cause no marked obstruction, for on account of the increased room we also find there is a tendency for the turbinate to recede in a compensatory manner.

Anteriorly, the same degree of deviation or presence of a spur becomes of marked importance the nearer it lies to the anterior meatus. Here the nasal process of the superior maxilla presents an unyielding resistance to any compensatory shrinking, and consequently all manifestations of obstruction become accentuated.

Hence it is that most operations on the septum have to deal with the triangular cartilage and of these the major proportion are confined to the cartilage itself.

Consequently, it is always worth considering any procedure involving the operation on these parts, which is calculated to facilitate the ease of operating, hasten the period of repair, and leave fewer sequelae.

Perhaps the simplest method proposed is to make a permanent perforation, removing in part or in its entirety,

the deviation by either a scalpel or so-called "punch forceps."¹

Next comes the method of incising the septum in one or several directions and with force attempting to bend the offending part toward the wider naris and hold it there till healing takes place. For example, the Asch², Rethi³ and Gleason⁴ operations.

A more elaborate method includes those operations which propose to remove part or all of the cartilage and bone involved without incising both mucous walls of the septum. To this method belong the procedures of Ingals⁵, Petersen-Hartmann⁶ and Krieg-Boenninghaus.⁷

In the Ingals operation only a triangular section of the deviated cartilage is removed, thus making merely a small channel of permeability for the obstructed naris.

The Krieg-Boenninghaus operation removes not only all the cartilaginous, but also all bony parts entering into the deviation. An incision of muco-perichondrium is made perpendicularly and horizontally on the anterior and superior lines of deviation, the same is now separated on both sides by means of an elevator and removed by scissors and forceps, with no attempt made to save the muco-perichondrium on side of convexity.

The Petersen-Hartmann operation attempts to prevent this sacrifice of the muco-perichondrium of the one side, which leaves a large surface bare of normal mucous membrane to be healed over with changed epithelium. A flap of muco-perichondrium is made, dependent from above and corresponding to the size of cartilage to be removed. This is allowed to fall into place after removal of cartilage and does in a measure reduce the area of denuded surface, but on account of the difficulty in making incisions and the large area exposed by shrinkage of the flap it is not in much favor.

Freer⁸ has suggested some modification in the manner of saving the muco-perichondrium and has devised a number of special instruments to assist in carrying this out. A triangular flap is made, the perpendicular incision being on crest of deviation, the horizontal joining this anteriorly on its lower end, and drawn forward on its line of hypothe-

nuse. Then with special knives a triangular area of cartilage is removed and through this window the remaining cartilage. Instead of removing what bony parts enter into deviation as in the Krieg-Boenninghaus operation, fractures by means of a chisel are made and these parts brought into alignment with Roe's forceps.

While I do not offer a new operation, I propose a different *point of one seen* for the operation, and attempt to save all of the muco-perichondrium, removing cartilage and bone by the Krieg-Boenninghaus method.

After the usual preparation of field of operation, cocaine the mucous surface with either a ten or twenty per cent. solution, injecting either a five per cent. solution or a Schleich solution into muco-cutaneous junction of septum. Adrenalin solution assists to keep field comparatively free from blood.

With an ordinary scalpel an incision is made perpendicularly along the entire anterior edge of the triangular cartilage or side of convexity, this being facilitated by drawing the fleshy part of septum sharply to opposite side. With a small, flat, somewhat curved on the flat, perichondritome or elevator the muco-perichondrium is separated over the convexity. Then beginning at anterior edge of cartilage do the same on side of concavity. The field of vision is improved by having the assistant, who stands behind patient to steady head, use one or two blunt hooks or retractors to open naris in place of nose speculum. If desired a stitch can be taken in fleshy portion of septum, which will include a strip of gauze. This will help to retract anterior edge of septum to opposite side. When both sides of cartilage are stripped the mucous membranes generally balloon to opposite sides and expose the cartilage in its entirety.

The cartilage is now cut out preferably with a pair of Grünwald's alligator scissors. If more is desired to be removed superiorly or inferiorly, it can be trimmed down with a pair of Krause's fenestrated cutting forceps.

Should there be a bony involvement in the deviation, continue the separation backward of periosteum, and remove the bone with a pair of fenestrated cutting forceps, preferably those of Laurent.

If deviation involves only posterior half of cartilage, the in-

cision in same can easily be made in front of convexity and muco-perichondrium separated only over concavity of deviation.

With all deviation removed, the linear incision can be stitched or not as suits the operator, while moderate snug packing on both sides brings the muco-perichondrial walls firmly together and prevents an accumulation of blood between them.

In order to utilize this incision so far forward for those deviations situated farther back on septum it is necessary to strip a part of cartilage which is not removed. But there is no danger in this as it immediately adheres to mucous membrane on being again coapted. Thus the incision is maintained well forward where it can be preserved from injury during operation and completely united afterward. It is in a position through which it is easier to work and allows of better control of instruments when working far posteriorly and a good view of field of operation.

Nor is its utility limited to deviation. All spurs on cartilage or on cartilage and bone can be removed without interfering with the intactness of the mucous membrane. Through this incision first separate the muco-perichondrium and then by means of a chisel, preferably a Hajek spur chisel, the spur can be cut off and removed through this incision by forceps. The mucous membrane is then coapted to the straight and smooth septum by packing the naris involved. Thus no subsequent crusting and granulation tissue has to be dealt with; for the nearer the anterior meatus the more frequently this unpleasant sequela accompanies loss of mucous membrane.

It is no longer necessary to defend the procedure of removing cartilage and bone of the septum. This has been done well in the papers of the authors of the various operations and experience has proven the theory. I would say, though, that it is scarcely ever necessary to remove a strip of cartilage lying in apposition with the external bones and cartilages of the nose, and farther, in the greater proportion of cases there is reformation from the perichondrium.

The following cases are illustrative:

CASE I: Frank W., aged 33. Large hemispherical devia-

tion of septum toward right side involving most of the triangular cartilage and with an offshoot, running onto vomer. Incision at anterior edge of cartilage and removal of deviation. Two stiches taken. Time of operation, including preparation and cocainization, fifty-five minutes. Packing removed on third day, none being replaced. Stitches removed on fifth day, when septum was perpendicularly straight in median line and with healthy normal mucous membrane on both sides.

CASE II: Charles S., age 12. Left nostril completely occluded with a cartilaginous deviation, while the anterior edge of cartilage projected one-third of an inch beyond the median line to right, drawing extreme tip of nose with it. Under cocaine anesthesia anterior half of triangular cartilage was removed, including upper anterior angle, both straightening the tip of nose and removing deviation. Time required forty-five minutes. In spite of the patient's youth, he stood the operation with scarcely a complaint till almost through, when a reapplication of cocaine allowed completion.

CASE III: Henry E., aged 35. Large horizontal spur or ecchondrosis of left side of cartilaginous septum, appearing in external meatus and extending backward over an inch, projecting outward across naris. Usual incision and separation of muco-perichondrium, and then, with a Hajek spur chisel, spur was cut off flush with balance of septum and removed with rat-tooth forceps. One stitch was taken in incision and small gauze packing inserted to hold muco-perichondrium in apposition with cartilage. Gauze removed on third day, stitch on fifth day, with a perfectly normal unbroken mucous membrane to be seen on a straight septal wall.

CASE IV: Peter T., aged 29: Unreduced fracture of nasal bones from injury when child, with resulting "saddle-back" nose. Deviation of cartilaginous septum completely blocking left naris. On right side of septum a large horizontal spur, involving cartilage and bone, beginning anteriorly where deviation originates and so large as to half close right naris.

Through the usual incision cartilage removed so as to entirely and thoroughly restore left naris. Then through same incision the perichondrium and periosteum over spur on opposite side was elevated and spur cut off with chisel and re-

moved with forceps. Both sides packed with gauze and no stitches taken. Time of operation fifty minutes. Packing removed on third day and replaced by fresh, which remained two days longer. On tenth day corrected "saddle-back" depression with paraffin injection, when the septum was shown with intact normal membrane and all nasal obstruction completely removed.

I think these cases are sufficiently demonstrative of the utility of this line of procedure in such septal operations to claim for it:

- 1st. Shortening of time of operation.
- 2nd. Better command of field of operation.
- 3rd. Less hemorrhage, both at time of operation and secondary.
- 4th. No flap that can be injured during operation.
- 5th. Preservation of entire intact mucous membrane.
- 6th. A resulting straight and even septum.
- 7th. Need of but few instruments, and these not necessarily specially constructed for this operation.
- 8th. Should mucous membrane be punctured on side of concavity, a perforation does not follow.
- 9th. Shortening of time of repair.
- 10th. Obstruction of narium satisfactorily removed.

¹O. Bergmann: Verletzungen. Fracturen, Dislocationen der Nase, Handb. d. Lar. u. Rhin. III.

²M. J. Asch: Trans. of 12th Annual Meeting of the Am. Laryngol. Ass'n.—1890.

Emil Meyer: "Deviation of the Cartilaginous Septum, its Cure." N. Y. Med. Jour., Dec. 1895.

Emil Meyer: "Asch Operation for Deviation of the Cartilaginous Nasal Septum." Med. Record, Feb. 1898.

M. J. Asch: Laryngoscope, Vol. VI, 1899.

³Rethi: "Die Verbiegungen der Nasen-Schildewand." Wiener Woch. 1890.

⁴E. B. Gleason: "Treatment of Deflection of the Nasal Septum." Jour. A. M. A., Vol. XXXVI, March, 1901.

⁵F. Ingals: "Deflection of the Septum Narium." *Arch. of Laryngol.*, No. 4, 1882.

⁶Hartmann: Partielle Resection der Nasen-Schiedewand bei hochgradiger Verkrümmung. *Der Med. Woch.*, No. 51, 1882.

Petersen: Ueber Sub-perichondriale Resection der Kuorpeligen, Naseschiedewand, No. 22. 1883.

⁷Krieg: Beiträge zur Resection der Cartil. Quadrang. Sept. Nar. zur Heilung der Scoliosis. *Berl. Klin Woch.* No. 31, 1889.

⁸Freer: "The Correction of the Nasal Septum with a Minimum of Traumatism." *Jour. A. M. A.*, Vol. XXXVIII, March, 1902.

XLVIII.

PLATINUM RHINITIS.

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The etiology of the various forms of rhinitis has been so exhaustively treated that at first thought it seems superfluous to mention another factor, but in the cases to be reported this element, hitherto I believe, unrecorded is so plainly evident and of such importance as to merit special consideration.

Mr. A. G., age 36, photographer, was referred to me in October, 1902, by Dr. H. J. Wetherill. He had always enjoyed excellent health and until two years ago never had any trouble referable to the upper respiratory tract.

At that time he began to have frequent attacks of what was diagnosed "Hay-fever" and the picture presented was typical; nasal occlusion, hydrorrhea, sneezing and lachrymation. Various local and general treatments were given without avail and the symptoms have persisted, without interruption and with varying degrees of severity, until the present time.

He has been a photographer for twelve years of which the past four have been almost entirely devoted to work with the popular platinum prints. In 1901, two years after taking up this particular paper, the above mentioned symptoms developed.

In the beginning the symptoms never appeared until he had been at work in the dark room for from thirty to ninety minutes and rapidly subsided upon reaching the open air, but at present they originate within a few minutes and never completely subside.

When handling other prints the attacks do not occur.

As they are called forth by use of the dry paper, the oxalate of potash, muriatic acid and phosphoric acid with which the prints are treated need not be considered, for he never suffers when the previously cut paper is immersed in the bath.

This seemed like a clear case of rhinitis vasomotoria due to platinum chloride, but to exclude any other factor a careful general examination was given with a negative result.

The urine was normal and there was no evidence of a uric acid diathesis, or of a neurotic temperament.

The mucosa of both nostrils was highly inflamed, water-soaked and sensitive, and the inferior turbinal of either side was in tight contact with the septum. Under cocaine and adrenalin they retracted to normal and the only permanent pathological condition found was a small spur upon the anterior, inferior edge of the quadrangular cartilage, right. There was an accompanying acute congestion of the epiglottis and mesopharynx.

Cauterization of the hypersensitive areas with the subsequent use of adrenalin and oleaginous sprays caused the symptoms to completely disappear for one month after which they gradually returned.

Henceforth the nose remained patulous, but the hydrorrhea, sneezing and lachrymation were intense after each exposure. The spur was then removed, more tissue lightly cauterized and internal treatment instituted with nothing more than partial temporary relief.

Nasal tampons were unavailingly used and resulted one day in so completely blocking the nostrils that he resorted to mouth breathing, with the occurrence that night of a severe attack of laryngeal edema.

This completely disappeared after two days but thereafter the use of tampons was abandoned.

No relief from treatment having resulted I advised complete abandonment of this paper since when there has not been a recurrence.

Two similar cases with almost identical histories have been seen and one of these reports that his partner suffers in the same way to such an extent that he is about to retire from the business.

A fifth case has recently come under observation which presents features analogous to the others, but differs in one important particular. While paroxysms are precipitated by handling the dry paper, they are also produced by the use of the platinum toning solution—with which the American Aristo paper is treated. This solution is composed of phosphoric acid and platinum chloride, and here the symptoms can be definitely ascribed to the platinum for while phosphoric acid may produce rhinitis it is of a different type from the one here seen.

These cases can be classed under the head of "Occupation Coryzas," or "Coryza Professionalis," but in the long lists of atmospheric and chemical agents reported as causative factors, I can find no mention of platinum chloride or of any special predisposition on the part of photographers.

We find recorded the vapors and fumes of such substances as potassium bichromate, chlorin, mercury, ammonia, arsenious acid, iodine, bromin, osmic acid, benzoic acid, ipecac, muriatic and nitric acids, sulphur, fluorine, copper and zinc, and substances that act through their constitutional effects like phosphorus, chromic acid, arsenic, cement, lime, iodides, cinchona and digitaline.

Such occupations as engineering, bronze workers, thrashers, spice grinders, chemists, apothecaries, coal miners, millers, workers in wood, either carving or planing, weavers, metal grinders, stone workers, etc., have long been recognized as especially predisposing to rhinitis, either simple or ulcerative, some acting through purely mechanical means and others by their chemical effects.

In the five cases which I have seen the condition produced was analogous to hay-fever, and the absence of any ulceration or cartilage necrosis would indicate that the platinum acts as a pure mechanical irritant.

While these cases present nothing of special import from the stand point of pathology or symptomatology, not varying in any respect from the picture presented by vasomotor rhinitis due to pollen or allied irritants, they are of considerable interest and importance in their etiology.

It cannot be absolutely affirmed that the platinum is solely at fault, for it may be that some other irritant sub-

stance is combined with the platinum in this paper. As the process of manufacture is secret I was unable to determine this point.

Taking the two facts, the rhinitis due to the handling of the dry paper and that caused by the platinum chloride in solution with phosphoric acid, a clear case seems to have been made.

When we take into consideration the vast number who follow this profession and the present tendency toward platinum prints, it would seem that this condition must be fairly common and my experience, five cases within the past year, lends weight to this relief.

I know of no effective procedure aside from change of profession or the discontinuance of work with this particular substance.

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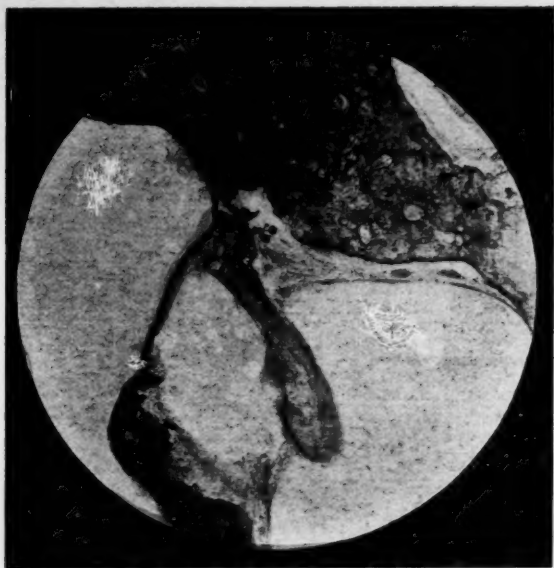


Fig. 1. Section through the anterior part of the oval window of the right ear of case 1. The bone superiorly is sclerotic and ulcerated both above and below, while the overlying periosteum is thickened. Magnification 25 times.



Fig. 2. Section through the same window further posteriorly, passing through the head, the posterior crus, the posterior part of the macula utricula, and the facial nerve. The bone above the niche of the window and the superior part of the base are diseased. Bony ankylosis. Magnification 15 times.



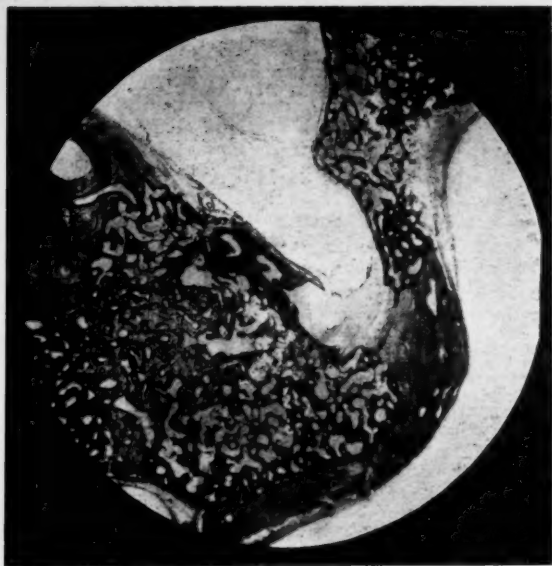


Fig. 3. Section through the promontory just before the niche of the window of case II, right ear. New formed bone in the scala tympani. Magnification 15 times.

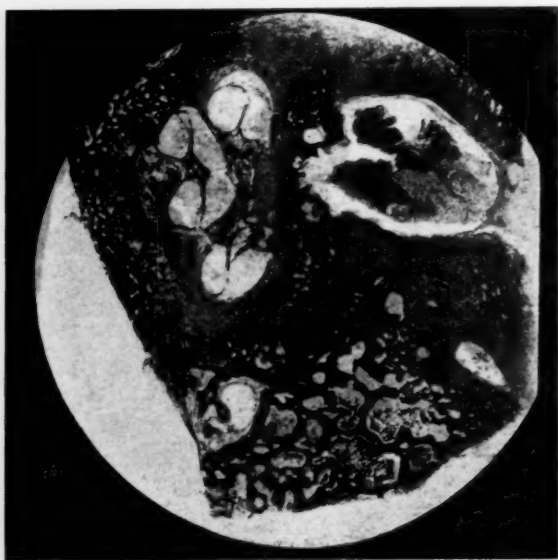


Fig. 4. Section through the anterior part of the cochlea and the internal meatus, case II, left ear. Small fragments in the apical turns of the cochlea. Disease of the bone at the anterior periphery of the internal meatus and the internal wall of the middle ear. Endosteal new formed bone in the scala tympani of the basal turn. Magnification 6 times.



Fig. 5. Section through the promontory, the facial nerve, the right vestibule and the end of the basal turn. The scala tympani partially filled out with bone. Magnification 9 times.

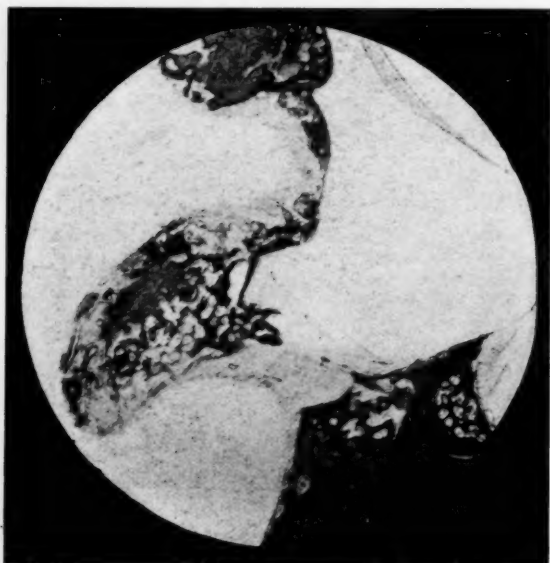


Fig. 6. Section through both windows and the macula acustica utriculi of the same ear. Case II, left ear. Ankylosis of the stapes. In both niches, considerable connective tissue. Magnification 15 times.

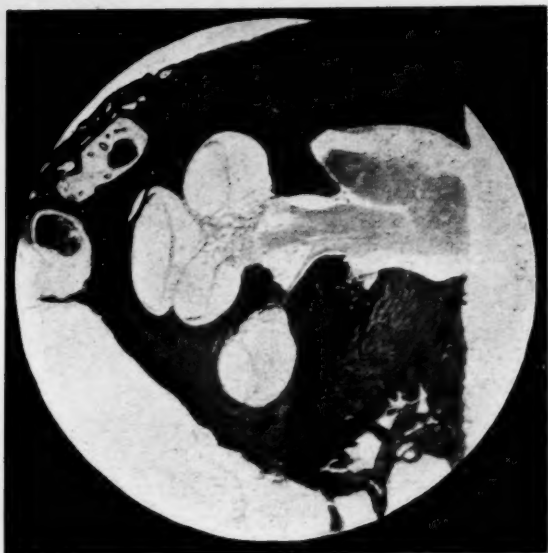


Fig. 7. Case III, left ear. Section through the cochlea and internal meatus. Lesion in the internal meatus. Magnification 7 times.

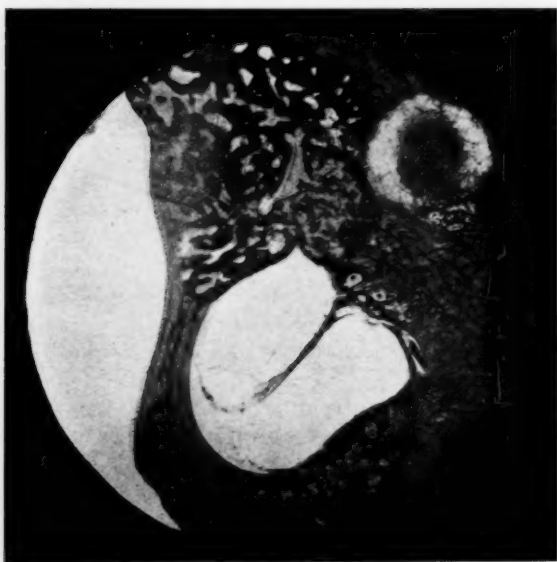


Fig. 8. Section through the promontory, the end of the basal turn and the macula acustica sacculi rotundi. Ostitic focus from the round and oval windows. Different stages of the ostitis near one another. Magnification 15 times.

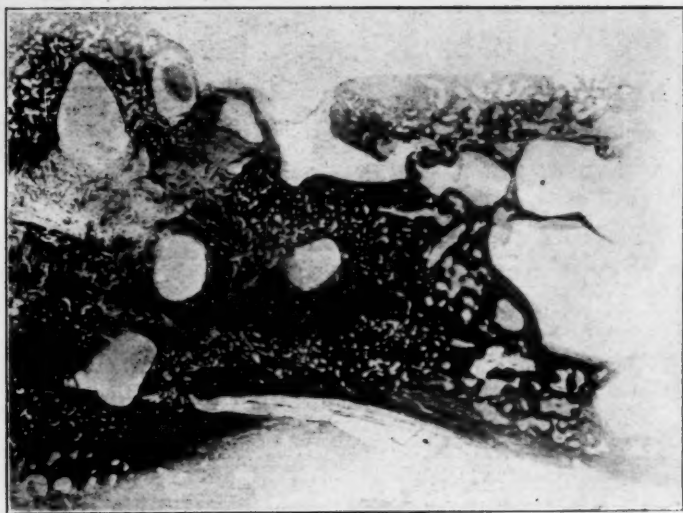


Fig. 9. Case VI, right ear. Section through the mastoid, the semicircular canals, the facial nerve, and the aqueductus vestibuli. Magnification 7 times.

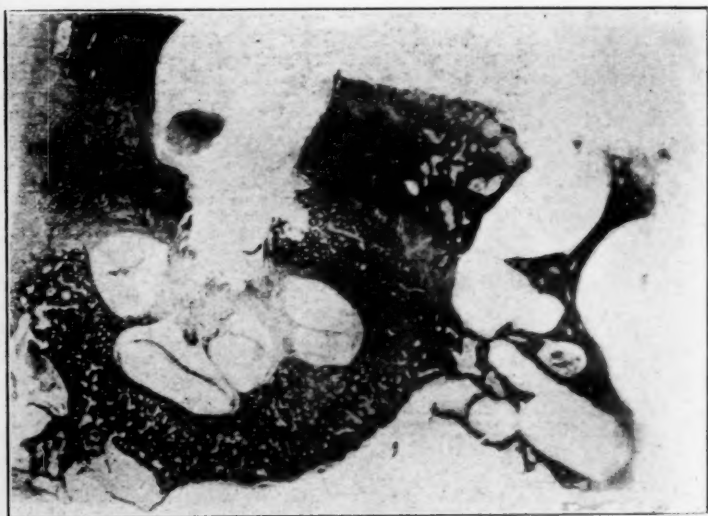


Fig. 10. Case VI, left ear. Section through the cochlea and internal meatus. The facial nerve is wanting in the section and the ligament, spirale was detached during the preparation from a part of the middle turn. Magnification 7 times.



Fig. 11. The same ear. Section through the window. Luxation of the stapes and an apparently isolated focus in the bone. Magnification 7 times.

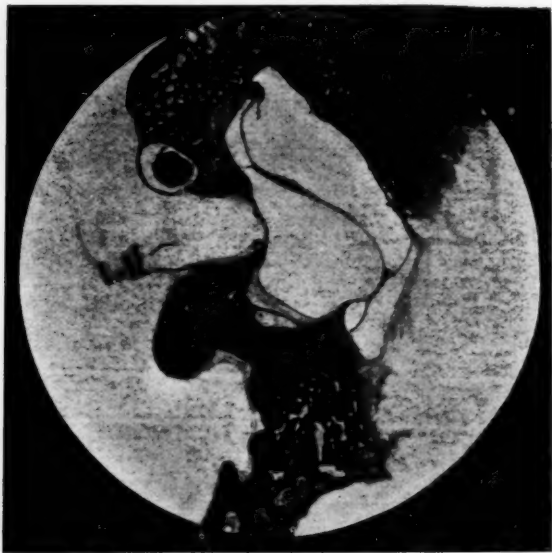


Fig. 12. The same ear. Section somewhat further posteriorly. Magnification 7 times.

XLIX.

THE PATHOLOGY* OF THE SO-CALLED OTOSCLEROSIS.*

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GRAZ.

TRANSLATED BY

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The so-called otosclerosis has been the subject of otologic investigation for years, but it is yet so hazy and indistinct, that it is still necessary to investigate it. My own experiences with this subject, which reach back to the year 1891,† and which are scattered in my article on pathologic anatomy in Schwartz's Handbuch, and partially reported in different otologic meetings, are now presented in toto as a contribution to the knowledge of this disease. I have delayed till now to do so, by reason of the fact that I am continually looking for further light from new material.

CASE I.

The first case of this disease that I had an opportunity to examine is described in the second division of "nerve atrophy in the internal ear,"‡ as the last case, with the diagnosis, "a recovered inflammation of the middle ear, and a cured otitis in the neighborhood of the base of the stapes, ankylosis of

*Archiv. f. Ohrenheilkunde. Bd. 60, S. 37.

†Zeitschrift f. Heilkunde, 1891. Bd. XII. S. 381.

‡Ebenda. S. 357.

the stapes and atrophy of the nerve on both sides, in the cochlea." As this report is not well known, I recall briefly the most important features, with certain amplifications, which I have lately found on again looking over the specimens.

C. Adalbert, 63 year old day laborer from G., had had impaired hearing for 20 years, so that he could understand only the loudest voice near the ear. As to the cause of his ear trouble, unfortunately nothing was known. He laid for a short time sick with icterus and chronic nephritis in the clinic of Prof. v. Jaksch in Prag, and died there April 10, 1890.

The post-mortem, which I reported *in toto*, gave no explanation of the temporal bone affection.

On examination I found in the right drum a large cicatrix with a cone-like incorporation of the epidermis, the lining of the middle ear without any important change, and in the anterior surrounding wall of the oval window, as well as in the external wall of the niche of the round window, a focus of chronic otitis of the pars petrosa, which was described as follows: "In the vicinity of the base of the stapes, just below the facial canal, beginning at the oval window and reaching downward to the upper border of the ligamentum spirale of the end portion of the basal turn of the cochlea, and backward and upward to the external border of the macula acustica utriculi, the bone was pathologically changed. This part of the bone was clearly distinguishable from the normal, and the border between the two was usually sharply defined. The altered bone accepted the hematoxylin and carmine stains much stronger than the normal. In the diseased bone the Haversian canals were plainly increased in size, as were the bloodvessels passing through them. In many places there were also to be seen cells, similar to osteoblasts, in a row and lying next to the bone. The bone itself showed a very irregular structure, the bone corpuscles were often very irregularly scattered, in places seemingly increased in number and the bone in many places resembled, more, calcified cartilage. Just over the stapes was a large hole in the middle of the bone, which was filled out only with connective tissue. Similar holes were found immediately beneath the deeper layers of the mucous membrane in the niche of the oval win-

dow, both over the stapes, and also, more extensive, beneath this, and there the bone showed cavities filled with connective tissue as far as the periosteal layers of the labyrinth. The stapes itself showed no defect in its base; on the contrary, presented extensive calcareous deposits, which reached from the labyrinthal wall upon the stapes and so caused a partial ankylosis of its articulation. In addition to this circumscribed ankylosis by calcification, there were calcareous deposits in the cartilaginous portion of the articulation and of the connecting ligaments. The joint and adjoining bone were normal only on the posterior, inferior periphery of the stapes. That the tissue adjoining the pathologically changed bone had taken part in the disease, was proven on the one hand by an extensive thickening of the connective tissue in the neighboring periosteal layer of the mucous membrane lining the middle ear, and on the other hand, by a similar thickening of the vestibular endosteum, which bordered on the otitic focus, which showed numerous spindle cells, and also calcareous deposits in places.

The left drum was thin, atrophic and without a cicatrix. The lining of the middle ear and the changes in the bone resembled those of the right ear, with the difference that there was no disease in the external wall of the niche of the round window, and the process at the oval window extended from the latter's posterior circumference into the bone as far as the point of the nerve's passage through to the external ampulla. The process had also attacked the posterior portion of the base of the stapes, and had caused a bony ankylosis. In the anterior half of the niche of the stapes was a large mass of fibrous connective tissue, which surrounded the anterior bar.

To this description of the bony changes, I must add, after having studied over the old specimens, that the changes caused in the bone by the disease showed a varied character. There was found, on both sides, at the anterior periphery of the oval window, a plain sclerosis of the bone, which in places showed calcareous deposits; toward the middle ear, there were foci where the bone had a more porous character and was penetrated by wider vessels and medullary canals; while on the anterior and inner borders of the diseased focus, in the

bone as well as in the posterior periphery of the niche of the oval window, the disease showed a more acute character. The bone there was colored better by eosin, its cells were larger, and it was more of osteoid bone, traversed by numerous vessel spaces, which contained large vessels, numerous large cells, and isolated giant cells (osteoclasts). According to these findings, I must correct the title of cured otitis used at the time I reported the case, in so far that this is true for only a large part of the diseased portion, since the process in the periphery of the focus was caught in the stage of progression.

At that time I considered the purulent otitis media as the cause of symmetrical affection of the bone of both ears, for this disease had certainly been present in the right ear at least, and I assumed that both ears were affected by the same cause, perhaps as the result of another acute disease, or as the result of typhoid fever. The high degree of deafness had lasted at least 20 years before death, and it is possible that the changes at the window had lasted equally as long.

CASE II.

Magdalena N., 61 years old, servant, came on May 28, 1891, to the II medical division, in a moribund condition. According to the history obtained from her daughter, the patient for 15 years had suffered with severe and increasing deafness and severe subjective noises in the ears. Pain was present at times, in the beginning, as were dizziness and headache. A discharge from the ear was never observed. For about 10 years the patient was almost entirely deaf; she understood her daughter only when the latter shouted loudly into her right ear. In the left ear, which always had been worse than the right, she could no longer understand her daughter. The dizziness and pain ceased with the coming of complete deafness, while the ringing and crackling persisted, but with intermissions. Otherwise she was always healthy, suffering only from toothache and headache. The cause of the ear trouble was considered to be overexertion, from nursing her very sick parents 30 years before. At that

time she frequently complained of trouble in the head. An examination of the ears could not be made *intra vitam*, as the patient had a high fever and was delirious. The post-mortem of the patient, who died on May 28, 1891, showed:

Body medium sized, greatly emaciated, of strong bony frame. Cranium correspondingly large, longitudinally oval, symmetric, rather thick and compact. The vessel grooves on the inner surface were shallow. The dura was stretched, smooth, rich in blood. The sinus contained a little clotted blood. The pia was smooth in its convexity, slightly clouded over the vessels which were everywhere well filled with blood. The white matter of the brain was soft, moist, and sprinkled with numerous small and large bleeding points. In the ventricles, there was a large mass of serous fluid, by which they were distended. The plexus hard, containing little blood. The central ganglia of the cerebrum rich in blood. The cerebellum of similar appearance to the cerebrum, also rich in blood and moist in all parts. Pia of the base of usual character. The sinuses filled with fluid, dark clotted blood, subcutaneous tissue rich in fat, muscular thin, red brown. Fluid blood in the jugular.

The heart lay obliquely, correspondingly large, rich in fat, containing clot; the cavities of normal size; the walls of proper thickness, reddish brown. The valves were normal. Intima of the aorta in places showed calcareous deposits.

The apex of left lung adherent by connective tissue, the upper lobe contained air except where the apex showed a small cicatricial contraction, and there were scattered through it isolated hard fibrous, pigmented foci; otherwise it was edematous and rich in blood. The lower lobe everywhere contained air, was rich in blood and contained more fluid than the upper, everywhere in the bronchi there was tenacious mucous. The apex of the right lung was adherent by connective tissue, similar in appearance to the left. The upper lobe had a focus which was large, tough, pigmented and contained calcareous salts. Middle and inferior lobes similar to the left. The stomach was unusually dilated. The umbilical ring immensely dilated and through it the peritoneum was bulged out into a hernia in which was included the entire large omentum, up to its insertion, the

whole pars pylorica, and the beginning of the duodenum. The former was fixed in the hernia, the latter were free and enclosed at their free ends by the border of the umbilical ring. The spleen was very small, the capsule slightly thickened, the tissue soft and friable, the pulp scanty. The left kidney of medium size, the capsule delicate, easily detachable, the tissue firm and rich in blood. The right kidney somewhat richer in blood than the left. The serosa of the greater curvature of the stomach injected with blood, showed numerous ecchymoses; in it was a cloudy, bloody colored fluid. On the posterior stomach wall were two ulcers, one lying over the other, the upper of which was somewhat wedge-shaped, 1.5 cm. long, and the inferior was somewhat rounder and smaller. The borders were soft, and the floor was covered with a mass like coffee grounds. Otherwise the mucous membrane showed stasis, ecchymoses and swelling. The mucous membrane of large and small intestines was injected and contents slight. The sigmoid flexure and rectum filled with scybalae. The liver of medium size, its surface smooth, its peritoneal covering slightly thickened. On section the tissue was of soft doughy consistency, very bloody, dark red colored. The gall bladder medium in size and filled with thick, dark brown gall. The bladder wall thickened, contracted and containing almost no fluid. Genitalia normal.

The pathologic-anatomic diagnosis was hernia umbilicalis incarcerata. Tuberculosis obsoleta pulmonum.

The examination of the auditory apparatus, which was given over to me, revealed nothing special on the drum or in the nerve in the internal meatus. The bone was not sclerotic and was comparatively easy to saw through. When the air in the internal meatus was compressed, a manometer attached to the superior semicircular canal showed no movement of the fluid. The mucous membrane of the middle ear and antrum was apparently unchanged, the stapes immovable in the oval window. The larger vessels on the promontory were filled with a large quantity of blood. The external part of the temporal bone was hardened in sublimate, the internal in Müller's fluid, decalcified and examined histologically.

MICROSCOPICAL FINDINGS.

RIGHT EAR—INTERNAL EAR.—A large amount of pigment between the nerve and the ganglion cells in the cochlea, along the lamina spiralis, in the stria vascularis, and a large quantity of free pigment also lay on the stria vascularis. At the end of the basal turn was found an extensive hemorrhage in the ligamentum spirale. The ganglion cells in the reverse portion of the basal turn were lessened in number, a large part of the scala tympani, the lowest turn, was filled out with new-formed endosteal connective tissue. A large part of the new-formed bone bordered on a chronically inflamed focus in the previous portion, and like this was pathologically changed. The periosteal covering of the vestibule was for the most part calcified, large quantities of pigment being deposited in and upon this, and, likewise, considerable pigment in the cristae and the neighboring portions of the superior and external semicircular canals, below the epithelium and between the nerves. The disease of the bone reached to the point of passage of the ramus vestibularis to the macula utriculi, but did not affect this. The internal wall of the membranous semicircular canals was mostly smooth. Only on the external portion of the external canals were found small prominences (papillae). The aqueductus vestibuli was free, the neighboring vein considerably dilated and full of blood; the aqueductus cochlearis was open where it empties into the cochlea, but could not be found further on in the desired bone, but its internal portion could plainly be seen. I was unable to find the vein of the ductus venosus.

MIDDLE EAR.—Only a portion of the bony Eustachian tube was in the specimen. The lining of the inferior wall was enormously thickened by increase of the submucous and periosteal connective tissue, and numerous villous projections were to be found on the superficial surface. The surface of the bone was often uneven, as though corroded, large vessel passages led to large cavities in the bone with similar surfaces, which were filled out by fibrous connective tissue. At many places, the walls of these cavities in the bone showed considerable calcareous deposits, as was to be seen by the more pronounced staining with hematoxylin.

THE DRUM, as a whole, was thin and only the mucous membrane layer, which consisted of fibrous connective tissue without distinct nuclei, was in places thickened. In the tympanic cavity the mucous membrane of the promontorium was somewhat thicker, and contained a rather large number of spindle cells and, in places, developmental cells, which could be followed, by means of the dilated vessel channels, into the bone. The mucous membrane of the inferior half of the promontory was especially thickened. Here was found a thick periosteal layer of sclerotic connective tissue, which contained only a few long spindle elements, and the thickened mucous membrane lay on this layer with evident signs of a chronic inflammation. The anterior portion of the niche of the round window was closed, partly by exostoses of the diseased bone, partly by fibrous connective tissue, but the posterior part was free. Yet, even here, on the inner side of the membrane of the window, was found the new formed bone mentioned above, which filled out the greatest part of the scala tympani. A plate of bone was formed which continually grew thinner, and which was found only in the center of the membrane in the sections through the posterior portion of the window. This bone was in direct communication with the diseased bone of the promontory. The membrane of the round window itself, was not thickened and was otherwise normal.

The anterior portion of the oval window was likewise narrowed by hyperostoses of diseased bone, and the base was entirely enclosed by it, which here extended into the vestibule in the form of a flat exostosis. An irregular calcareous mass, without evident bone cells, surrounded by thickened connective tissue, lay here in the diseased bone. Further posteriorly, the base was plainly seen, but, especially on the inferior side, to far posteriorly, was in bony union with the diseased bone, and the inflammation reached into the anterior part of the base of the stapes. Only the posterior part of the base of the stapes and its vicinity was free from disease. The crura of the stapes were thin, as was the base, the bone alternated and the articular facets were not present. The cartilaginous surfaces of the hammer-anvil articulation were for the most part calcified.

The mucous membrane of the ANTRUM and MASTOID as well as that of the posterior part of the tympanic cavity and aditus were similarly thickened and altered. In places, signs of chronic inflammation and thickening with spindle and developmental cells, especially in the dilated vessel channels of the neighboring bone, and often in the latter were cavities filled with connective tissue.

BONE.—In the anterior inferior portion of the temporal bone were enormously dilated medullary spaces filled with medullary substance, and between them pneumatic cells lined with thickened mucous membrane, and some cellular spaces entirely filled with connective tissue. Above these and in connection with them, the bone was pathologically changed as far as the anterior inferior periphery of the cochlea and the anterior inferior wall of the internal meatus. It was traversed by large vessel spaces, in places, near the border, sclerotic, and showed in the wall of the internal meatus several large cavities, which were partially filled with connective tissue rich in cells which was in direct communication at this place with the periosteum, similarly diseased, thickened and rich in cells. The bone in these spaces, as well as elsewhere in the internal meatus, was uneven on the surface as though eroded (Howship's lacunae). They are also, in connection with the vicinity of the canalis ganglionaris and the bone of the lamina spiralis of the basal winding, calcified, as is seen by the intense coloring with hematoxylin. The inflammatory focus in the bone reaches as far as the periosteum of the basal cochlear turn. It attacks the portion that lies below and extends forward, in its inferior, internal and partially external part, and reaches here nearly directly the periosteal coat of the ligamentum spirale. A large part of the basal turn, as a result of the inflammation in the bone, is affected with new-formation of connective tissue and bone which fills out most of the end of the scala tympani as far as the membrane of the round window. This new-formed periosteal bone is later attenuated by the chronic inflammation of the bone in the petrous portion and fresh inflammatory changes are found, as in the surrounding bone. Posteriorly, the focus reaches to the round window in whose anterior portion the diseased bone forms exostoses both on the promontory and

the inner wall, while in the posterior part of the round window, the bone is nearly normal. Only the vessel channels of the bone contain richly proliferated connective tissue, and are surrounded by bare spaces of late growth.

A second focus of altered bone is found above the first on the inner wall of the middle ear. It reaches forward to the region of the apical turn of the cochlea, and here lies between mucous membrane and cochlear periosteum. From above downward it is from 4 to 5 mm. in height, and reaches upward to the facial canal and downward to the middle of the promontory. Internally, it borders on the apex, then on the middle turn, and further back on the vestibule, then surrounds the greater part of the stapes and reaches to the point of passage of the nerve from the utriculus through the bone, which is surrounded at its anterior external portion, and then ends at the posterior portion of the oval window, without reaching the latter's posterior border. Both of these diseased foci in the promontorial bone are joined only by a dilated vessel space, whose wall appears greatly calcified, and are otherwise separated, in the middle of the promontory by a layer of healthy bone.

Finally, a third focus of greatly altered bone is found between the facial and the external semicircular canal. It is sclerotic, traversed by medium dilated channels, in whose neighborhood, the bone is deeply stained by carmine and eosin. In addition, the other parts of the pars petrosa and mastoid show distinct changes. In many places the channels are obliterated, and even in the bone are found new-formed younger layers surrounding large remnants that stain deeply with hematoxylin. The former are sharply differentiated, by their peculiar lammellating layers, and clear red color, from the parts previously described. Large bone corpuscles with few projections and distinct cells distinguish this bone from the old.

The histologic character of the pathologically altered bone is different at different places. Ordinarily, the border portions of the diseased bone show the plain picture of sclerosis of the bone. The bone stains darker with hematoxylin, its vessels spaces, with the exception of isolated smaller ones, are obliterated by layers of new-formed bone along their

walls and often by deposits of calcium salts in the remains of the former lumen.

This part of the bone contains no medullary spaces, the bone cells are pressed close together, show numerous irregular projections and often on their inner wall are stained deep blue or black with hematoxylin as a sign of a more pronounced calcareous deposit. In places, a similarly affected bone lies next to this sclerotic bone, which however has less calcareous deposits, and contains numerous widened vessel spaces at other places in the diseased portion, and in the outer boundaries of the foci, especially at the inner wall of the promontory, the bone is altered in another manner. The bone shows lamellar structure, absence of the nests of cartilaginous cells otherwise present, stains light blue with hematoxylin, although not exactly like normal bone. Its bone corpuscles are in places larger, show fewer projections and plainly staining bone cells. This bone is traversed by large spaces. That contain scant connective tissue and isolated fat cells. In places in this bone, are parts that show a redder color, internally passing over into parts that stain deep red with eosin, in which the inflammation still persists. Here the bone is extremely porous, is traversed in all directions by extremely dilated channels, between which are small bridges of osteoid tissue. This never shows a lamellated structure, its cells are clearly larger and have fewer projections. Along the dilated channels lie long spindle cells, and large round cells with large nuclei. The latter are often found in the smallest lymph spaces of the bone which lead from the diseased into the neighboring healthy bone. They resemble osteoclasts which usually lie alone on the walls of the spaces leading into the larger vessels. Where they lie against the bone is usually a rounded space surrounding it like a semicircle. What role is played by the cells lying next to the healthy bone cannot be determined, since the healthy bone shows no changes up to its very border. As to the relation of these freshly diseased portions to the other bone, it can in general be said only that they lie further inward, further from the middle ear. Thus, fresh bone disease is found at the anterior inferior border of the cochlea, likewise corresponding to the apex of the cochlea, and at the posterior and

internal border of the focus at the oval window. The disease in the niche of the oval, and the anterior part of the round window shows occasionally a comparatively fresh character and here the small osteoid bands of bone which stain faintly with eosin reach to the periosteum of the middle ear which is greatly thickened, poor in nuclei and of a fibrous character.

LEFT EAR.—INTERNAL EAR.—The nerve cells in the cochlea, especially in the upper windings are not as numerous as under normal circumstances. The richness in pigment is somewhat less than the right. In the basal turns, a vessel in the *prominentia spiralis* is unusually wide. The *lamina spiralis* is unusually wide. The *lamina spiralis* and the surrounding of the ganglion canal of the basal turn are greatly calcified. In the *scala tympani* of this turn was newly-formed bone and connective tissue in large amount are found. The *ductus venosus* of the cochlea is free, the *acqueductus* in its inner portion likewise, but in the outer part is not demonstrable in the diseased bone. The vestibule and semicircular canals show the same condition as the right.

TUBA EUSTACHII.—The epithelium of the cartilaginous portion is well retained, and below this, in places, long portions of the mucous membrane are infiltrated and inflamed, epithelioid and spindle cells being especially prominent. Most of the lining of the bony portion was thickened, in places thinner, and contained cells in only a few places. The neighboring bone showed irregular jagged cavities filled with a dense fibrous connective tissue. The periphery of the bone in these cavities is usually very calcareous, and stains a dark blue with hematoxylin. Near the middle ear, in the medullary spaces of the vicinity of the tube, are profuse hemorrhages; in certain parts of the bone, filled with connective tissue, are large masses of calcium salts, and furthermore, certain jagged protuberances of the bone into the tube, resembling osteophytes.

The **DRUM** is unusually thin, and stains badly, so that formed elements are hard to distinguish. Irregular spaces, corresponding to the inferior border of the drum are found in the mucous membrane, whose submucosa is formed of fibrous connective tissue. Large numbers of similar spaces

are found in the mucous membrane covering of the hammer and anvil, representing the ligaments. No epithelium is demonstrable on their inner surfaces, the connective tissue of the submucosa is to a large extent calcified. On the hammer, internal to the short process, is a large space filled out with fibrous connective tissue. The bodies of the hammer and anvil contain numerous medullary spaces filled with medullary substance and dilated vessels. The articular cartilage is almost entirely calcified. Irregular masses of lime salts are found in places in the vessel channels and bone of the anvil.

MIDDLE EAR AND WINDOWS.—The mucous membrane of the tympanic cavity is almost everywhere changed. This is especially visible in its deepest layers, especially thickened over the diseased bone, even being callous, and it contains over the foci of acute bone disease numerous cellular elements, rows of spindle cells, while the well developed epithelium often follows immediately over this layer. At other places is a submucosa with wide vessels, spindle and developmental cells. Especially thickened is the mucous membrane in the anterior part of the niche of the round window, whose inner half is entirely closed by connective tissue. The oval window has somewhat less. In the latter are comparatively fresh inflammatory changes.

The articulation of the stapes with the oval window is ankylosed, partially by a more or less broad new formed spur of bone, which jutted forward from the surrounding diseased bone into the base, and partially by a calcification of the annular ligament. The ring itself is greatly attenuated by the disease of the bone and is actually defective at one place in the posterior part, so that the thin mucous membrane lies directly upon the periosteum of the inner wall of the vestibule. The latter as well as the subjoining cartilage, from which it cannot be separated, is calcified. In the anterior part of the oval window the diseased bone projects into the vestibule in the form of an exostosis.

The round window is free in the anterior portion, in the direction of the middle ear, but here the membrane lies on new-formed bone which fills out the greater part of the scala tympani. Further back this bone gradually lessens, and the

disease of the bone passes over upon the stapes, whose base is similarly diseased, thence to the neighboring bone of the promontory as far as the round window, which it posteriorly attacks and narrows by small exostoses from the side. Here fibrous connective tissue lies on the membrane of the round window, externally, and fills out the posterior portion of the niche of the window. In the anterior, internal part of the niche considerable pigment is imbedded in a looser connective tissue.

ANTRUM AND MASTOID CELLS.—The mucous membrane has the same appearance as in the middle ear, except that it is somewhat thinner, and thickened only in isolated pneumatic cells. The subjoining bone often shows lacunar formation, in other places large spaces filled with connective tissue, and often there are evidences of new formation of bone, beautiful areas of new-formed bone in vessel channels of the bone bordering on the antrum.

BONE.—The bone tissue of the temporal shows several foci of disease, which, with slight exceptions, have a similar distribution to that of the right side. In the anterior part of the pars petrosa are very dilated medullary spaces, and isolated, large pneumatic cells, and the latter are almost entirely filled out with connective tissue. The latter, which are connected with the middle ear, lie directly under the diseased bone, which surrounds the internal meatus on its anterior periphery, and forms a large exostosis in it, which rests on the nerve, and large spaces, partially filled with connective tissue, which are joined to the internal meatus. This focus borders for quite a distance on the lowest portion of the scala tympani and is in communication with the bone and connective tissue which fills this out. Posteriorly it reaches only to somewhat in front of the external mouth of the aqueductus cochlearis, in vicinity of which the bone is still healthy. The bone that partially fills out the scala tympani reaches somewhat further posteriorly than the otitis focus does. The second focus, in the vicinity of the stapes, reaches further forward on the left side, to the bony tube, and borders internally on the upper and middle turns of the cochlea, and externally directly on the periosteal layer of the lining of the middle ear. Further posteriorly, it

stretches, both above and below, the facial canal, which runs from the internal meatus to the middle ear, far inward, below nearly to the internal meatus, making a small exostosis in the canal itself. Posteriorly this focus stretches unusually far, to behind the region of the ampulla of the external semicircular, where it affects the entire bone between the facial canal and the external wall of the ampulla and the semicircular canal. This focus possesses a width of 5 mm. from above downward in the bone of the internal superior wall of the middle ear, between periosteum and cochlea, vestibule and external ampulla. It surrounds the base of the stapes, and the external portions of the branches of the nerve to the utriculus and the external semicircular canal at their passage through the bone. Its posterior part affects the entire promontory, to its lowest part, and here reaches to where the bone fills out the scala tympani, and also affects the posterior border of the round window's niche, while the bone of the anterior is still healthy. The base of the stapes is likewise totally diseased and in places forms a mass, which is connected with the labyrinthal bone. Only on the superior and inferior border are remnants of the annular ligament visible. These two foci have no direct connection with each other, yet there is found in the otherwise healthy bone in the inferior half of the anterior portion of the promontory dilated vessel spaces, with areas of new formed bone. Wide channels, with dilated vessels, run from the periosteum and above mentioned cells in the anterior inferior part of the pars petrosa into the diseased bone.

In addition to these two large foci the bone shows in other places, especially in the superficial subperiosteal layers, thin sheathes of osteoid or new bony tissue, especially in the region of the often dilated channels. The surface of the bone is corroded in places and contains irregular excavations.

The histologic character of the left diseased bone corresponds entirely to that of the right temporal. Likewise, the division of the bone by the past and the still present otitis, is entirely as on the right side, so I can omit any further description here.

If, in conclusion, we glance over the changes found in both temporal bones, as well as those in the lining of the middle

ear as in the bone, we must come only to the conclusion that we are dealing with the sequelae of an inflammation, which has passed away in the lining of the middle ear and partially also in the bone, but which still persists in places in the bone, or has recently become worse. We must believe, according to the history, that it began about 30 years before the death of the patient with severe ringing, and occasional sticking pains and deafness. This inflammatory process, then, in various places, passed over to the bone, doubtless anteriorly from the lower pneumatic cells upon the anterior border of the cochlea and the internal meatus, and then from the niche of the windows to the neighboring bone in which it caused a chronic otitis and osteomyelitis. It is plain that thus a large part of the end of the scala tympani was filled out with new-formed bone and connective tissue, and it is no longer possible to determine from the specimens whether this took place by a continuance of the process from the first described foci, or at the beginning of the disease, a severe otitis media passed over through the membrane of the round window to the end of the scala tympani and there caused an endosteal new-formation of bone and connective tissue. The latter is contradicted by the fact that this bone in the scala tympani borders only slightly on the membrane of the round window, and reaches its greater width further on. The extensive changes in the lining of the middle ear as well as the bony portions of the temporal bone bordering on the middle ear indicates an extensive inflammation of the lining of the middle ear at least originally present.

CASE III.

Alois St. 58 years; deaf since middle age. The hearing has been growing worse year by year. He has never had pain in the ear or dizziness. Often had dumb feeling in ear, and on the right side ringing and sounds like the striking of the clock. Often suffered from severe colds.

Examination of ear revealed the drum somewhat retracted and slightly milky and discolored; the light reflex clear. Left, drum greatly retracted, milky, discolored, the light reflex

dull. The examination of the nose and throat of the patient who lay in the I. medical division, was not made.

The tests for hearing gave:*

$$\begin{array}{c} W \\ R < L \\ O \left\{ \begin{array}{c} U \\ U_s \\ W_w \end{array} \right\} O \\ 1.0 \text{ St. } 0.50 \\ 0.02 \text{ Fl. } O \\ 7'' \text{ Cw } 6'' \\ - R - \\ 4'' \text{ c } O \\ - 20'' \text{ c}^4 - 25'' \\ - C^7 \text{ H}^\dagger \text{ C}^7 \end{array}$$

The patient died August 7, 1891, and the postmortem held Sept. 8, in the pathologico-anatomic institute gave: Well nourished, strong individual, edema of lower extremities. Severe edema of pia, much fluid in ventricles, whose ependyma is delicate. The vessels at the base of the brain rigid, the brain substance atrophic, very moist, medium rich in blood, narrow cortex. Free fluid in pericardium and pleura. Heart very large, muscle fragile, pale and yellowish spotted. Left cavity likewise wide, walls thick. The aortic valves, as well as those of the right heart delicate, the free border of tricuspid thickened, the chordae tendinae retracted and thickened. The lungs everywhere contain air, are rich in blood and very edematous, pressed together at bases. Bronchial mucous membrane dark red. The spleen large and indurated. Left kidney enlarged, capsule adherent, surface delicate, granulated. Pyramidal markings distinct, tissue hard, brownish yellow. Right kidney very small, deep infarcts, cysts and granular surface.

*W = Weber, U = Watch, U_s = watch on the Temple, W_w = watch on mastoid. St. = voice, Fl. = whisper. Cw = small Lucæ fork on mastoid (normal 16 seconds). R = Rinne (normal = + 36") c = the same directly after being struck, normally heard 56". C^4 = normally 42." H = Range of hearing for all forks by air conduction.

†The lower border not determined. C^8 not heard.

Gastric mucous membrane swollen and folded, very red and ecchymotic over the folds. Same symptoms of stagnation in intestines. Liver changed into form of stagnation, nutmeg liver. Mucous membrane of bladder delicate and pale, genitals normal, aorta atheromatous.

Diagnosis—Hypertrophia et dilatatio cordis totius praecipue cordis dextri cum degeneratione adiposa. Insufficiencia valvularium tricuspidalium. Venostasis et hydrops universalis. Atrophia renis p. infarctus. Atheromatosis aortae. Induratio.

At the post mortem, both middle ears and semicircular canals were opened, the temporal bones removed and hardened in Müller's fluid. Previously cultures were made from the profuse mucous contents of the middle ear. Those from the left middle ear were sterile; those from the right showed four colonies of *aspergillus glaucus* to the tube. During further section of the temporal bone, I found the bone around the antrum and in the mastoid very pneumatic, the stapes immovable, and the round window closed by connective tissue. The specimens were then decalcified, embedded in celloidin, and the histologic examination undertaken.

MICROSCOPIC FINDINGS.—RIGHT EAR.—The fundus of the bone of the internal meatus showed thin layers of new bone containing a large amount of calcium salts, as it stained very deeply with hematoxylin. Large hemorrhages in the fundus of the internal meatus.

The ganglion cells of the cochlea, especially the basal turns, greatly decreased, as are the nerve bundles in the lamina spiralis ossea. Nothing of importance in the vestibule and semicircular canals.

MIDDLE EAR.—The submucosa of the cartilaginous tube fibrous with few cells. At the isthmus is an anomaly, where there is a saccular bulging of the bony tube external and superior to the end of the cartilaginous, so that the superior wall of the cartilaginous does not pass directly over into the superior wall of the bony tube, but the latter lies several millimeters higher than the former. The wall of the bony tube shows projecting pieces of bone, between which deep excavations are seen. Several neighboring cells are filled with connective tissue containing spindle cells.

The lining of the tympanic cavity is in general somewhat thicker and of a more fibrous structure, with few cellular elements, the epithelium is well retained. In places, masses of calcium salts are imbedded in the mucous membrane. The lining is thickened especially where it borders on the diseased bone, and in both of the window niches.

The oval window is somewhat narrowed through increase in the surrounding diseased bone, and the diseased bone has entirely grown around the anterior part of the base of the stapes. A very thick fibrous connective tissue lies on this bone in which is enclosed the middle part of the anterior bar of the stapes. There is no stenosis of the middle and posterior part of the niche, since here both above and below, the superficial bony layers of the wall of the niche, with the exception of a few small remnants imbedded in connective tissue, have entirely disappeared on account of the disease, and are replaced by connective tissue. At the inferior posterior border of the niche, a large portion of the bone, up to nearly the vestibule, has been ulcerated away and replaced by connective tissue. The base is fixed in its anterior part, and somewhat outward, and cartilage and annular ligament form a calcified mass, which is connected with the diseased bone. Further posteriorly is a simple calcification of the cartilaginous layer, and only postero-superiorly is the calcified cartilage of the base firmly united with the diseased bone of the wall of the niche. The base itself consists in its anterior part of sclerotic bone, and in its posterior part shows no change other than a calcification of the cartilage continually increasing in amount. An exostosis narrows the anterior portion of the niche of the round window. and this, the further posteriorly it goes, gets even closer to the diseased medial wall of the promontory whence it springs, and reaches inward so that more and more of the membrana tympani secundaria are visible. The posterior part of the niche is completely free.

Only the mucous membrane of the drum is thickened, and considerable pigment grains are deposited in this as also in the layer of circular fibres. The chain of ossicles show calcareous deposits in the articular cartilage between the hammer and anvil, as well as between the latter and the stirrup.

The mastoid is very pneumatic, and the mucous membrane in aditus and antrum considerably thickened, but is not much altered in the cells.

The small arteries in the facial canal, the promontory and internal meatus show circumscribed thickenings of the intima due to endarteritis, and the latter also thickening of the adventitia, consisting of sclerotic connective tissue, poor in cells.

BONE. In the pars petrosa are three circumscribed disease foci. The first and smallest lay in the internal meatus on its inferior periphery, some millimeters from the bottom of the meatus, inward immediately below the thickened dural covering, beneath which the superficial bone is somewhat defective, connective tissue taking the place of the bone. It extends like a tongue downward consisting throughout of sclerotic bone with irregularly lying bone corpuscles and scant, somewhat dilated vessel channels which contain a fibrous connective tissue poor in cells, and into which emptied the channels of the surrounding and likewise sclerotic bone, for the most part obliterated and calcified. Otherwise, the bone showed sclerosis for a considerable space around this focus, as well in the periphery of the internal meatus, as further downward. Its canals everywhere stain deeply with hematoxylin and show neither lumen nor vessel. Forward and inferiorly, this bone borders on a large wide pneumatic space, which extends from the tympanum deep into the bone.

A second focus, which surrounds the oval window reaches with its apex forward to the middle turn of the cochlea, at whose periphery just at the ligamentum spirale, it ends. Posteriorly it becomes broader and surrounds the space between the facial above, the round sacculus inward, and the tympanic cavity, outward, and reaches posteriorly to the oval window whose periphery it surrounds, but the inferior border ends in front of the posterior border of the niche of the window, while the superior border extends in the bone as far as the ampulla of the external semicircular canal. This focus also shows, with few exceptions, throughout, old changes that have already ceased. It is traversed by wide channels in which a small amount of connective tissue with fat cells lie. In the periphery of this focus, are many scleroses of the bone, and the diseased bone everywhere is

distinguished by its structure and deep reddish blue color (hematoxylin-eosin) from the old, as new formed bone. Fresh inflammatory changes are found only at the anterior and posterior pole of this focus, and at the latter place, both above and below the niche of the oval window. Here the now-formed bone consists of osteoid tissue with osteoblasts, traversed by large channels with dilated vessels and large developmental cells. Giant cells, and osteoclasts demonstrable at the border of the healthy bone.

A third focus is found in the vicinity of the round window. It extends forward to the region of the external mouth of the aqueductus cochlearis, without reaching to the endosteum of the cochlea, then becoming wider surrounds the anterior border of the niche of the round window, and then especially on the inner side of the promontory, extends inward and upward into the scala tympani as an exostosis. This focus reaches only the external periphery of the round window, i. e., in the promontory, and in the posterior part of the niche is connected with the above described second focus at the oval window, as both hang together by a dilated channel with pathologically altered bone in its walls. This third focus is distinguished from the former by the fact that it shows almost throughout its entire extent fresh ostitic changes and only at places on the periphery sclerosis of the bone. The cellular infiltrated channels of this bone often border directly on the healthy bone.

The bone of the temporal bone shows almost everywhere pathologic changes. The cells which are ordinarily found in the bone of the pars petrosa above and below the labyrinth capsule, are very much reduced in size from within by deposits of new bone, and this is to be distinguished from the old only by its deeper staining with eosin and its different arrangement in layers. The channels in the bone of the pars petrosa have their inner walls more or less calcified and often entirely eroded. Likewise radical changes are found in the external portion of the temporal bone, in the vicinity of the tube, and the Glaserian space. The latter shows very large deposits of chalk on its bony borders, and the cells in the neighborhood of the bony tube are entirely filled out with fibrous connective tissue, and the surrounding bone is sclerotic.

The bone of the mastoid is comparatively unaltered. Only around the semicircular canals are sclerotic parts to be found in the bone, while the posterior parts of the mastoid are entirely pneumatic, without special changes.

LEFT EAR.—The INTERNAL EAR shows similar changes to the right.

MIDDLE EAR.—In the Eustachian tube, otherwise similar to the right is the same anomaly, only more pronounced. The end of the bony tube, in its superior portion extends somewhat further forward over the isthmus.

The lining of the middle ear is in general somewhat thickened and lumps of chalk are deposited in it in places. The inner layer of the drum shows considerable pigment. The anterior portion of the niche of the oval window is filled out by a rather thick exostosis of bone and further down and behind by fibrous connective tissue. Posteriorly, the lining is very thick, and is partially connected with fibrous connective tissue at the spots of ulcerated bone, and this encloses several remains of bone in which considerable chalk, but no plain bone structure, is demonstrable. The connective tissue mass shows itself on the inferior border to a large extent greatly calcified. The bars of the stapes are fixed to the wall of the niche by connective tissue adhesions, especially the posterior bar, while the anterior is partially enclosed in the exostosis. The base of the stapes in its anterior superior part, is largely dislocated out of its articulation with the labyrinth wall, and is joined to the neighboring wall of the niche anteriorly by new formed bone that has affected half of the base of the stapes, and further posteriorly and superiorly by strong connective tissue. The cartilaginous sheath of the articular facets and the annular ligament are greatly calcified and the chalk in the ligament can be followed into the upper border of the window as far posteriorly as the middle, where the articulation again shows a normal condition. In the most superior posterior part of the niche, the disease of the neighboring bone again passes over to the base of the stirrup, which here is again bound by bony union to the neighboring parts. The anterior portion of the niche of the round window is considerably stenosed by a large exostosis from the promontory and a smaller one from the medial wall, and the remaining portion

of the border of the niche is filled out by loose connective tissue. Posteriorly the niche is free, only somewhat narrower, because of the exostosis of the promontory, which becomes smaller the more posteriorly it goes, and the membrane of the round window is made appreciably smaller by this exostosis. The bone of the large ossicles is sclerotic, and shows abundant chalk deposits as do the cartilage of the short process and the articular cartilages of the hammer and anvil that touch the bone.

The mastoid is extremely pneumatic, like the right, and in it are also found large pneumatic cells at the posterior region of the aqueductus vestibuli. The mucous membrane in the cells is somewhat thickened, individual small cells in the anterior portion entirely filled out with connective tissue.

The arterial vessels of similar appearance to right. The bone of the pars petrosa shows 3 foci of disease similar to the right. The first, in the internal meatus, lies somewhat nearer to the cochlea and reaches into the bone of the labyrinth capsule itself. It consists of greatly calcified bone with dilated channels without acute inflammatory changes and is joined below by a piece of sclerotic bone with the cells beneath the cochlea, which here reach far inward and upward. The extent of the second focus corresponds almost exactly to that of the right side. It extends forward to the posterior superior border of the middle turn of the cochlea, but anteriorly is considerably smaller than the right, and the fresh otitic changes, in comparison with the old, reach a greater extent, since on the left side not only the anterior and posterior poles of the focus, but also the entire inner zone, as well as the anterior and posterior periphery of the niche of the oval window are freshly diseased, and at these places the disease is everywhere advancing toward the healthy bone.

The third focus, at the round window, is at its anterior periphery somewhat broader than the right, extends into the scala tympani in the form of a spongy exostosis, and is then confined to the medial side of the promontory, to end here at the posterior wall of the niche. Anteriorly it shows fresh otitis at the external and internal periphery, sclerosis of the bone, and posteriorly only more sclerotic bone with slightly dilated vessel channels. On the left side the vessel connection

in the promontory between the second and third foci is absent. What was said of the right side applies to the rest of the bone.

If, in summing up, we review the changes in both temporal bones, which for the most part are very similar, we must draw the conclusions from the diffuse changes of the lining of the middle ear, from the chalk deposits therein, from the filling with connective tissue of certain cells bordering on the middle ear, tube, and aditus, as well as from the diffuse changes in the bone, that we are dealing with the sequelae of a preceding otitis media, which extended from the tube to the mastoid and affected only the most posterior portion of the mastoid to a medium degree. The circumscribed foci in the bone of the pars petrosa can be considered only as the result of an extension and a continuance of this inflammation in these bony foci. This is strongly supported by the appearance of the focus around the stapes of the right side, in which can plainly be observed the progress of the ostitis from the anterior portion of the niche, forward to the periphery of the cochlea, and backward to the crest of the external semicircular canal. Doubt as to the origin of this focus by continuity of the inflammation from the lining of the middle ear can arise only in regard to the focus in the wall of the internal meatus. Yet since similar pathologic sclerotic changes are found in the bone between this focus and the wider pneumatic cells connected with the tympanic cavity, at the anterior inferior periphery of the cochlea, the continuity of the inflammation from these cellular spaces to the bony wall of the meatus is likewise possible, although I must assume from the histologic picture that it is probable that this focus took its origin from the periosteum of the internal meatus.

That all these foci existed at the same time, at the beginning of the disease, can no longer be proven; at least so far as the foci in the niches of the windows, it seems more probable that there were later exacerbations of the process. The findings at the oval window speak for a long continued, superficial ulcerative process at this place.

According to the history, the disease began about 30 years before the death of the patient, and then grew gradually worse. The explanation for this increasing deafness is plainly

found in the changes in the niches of the windows, especially in the stapes and its articulations, in the bilateral changes in the anterior portion which are old and have ceased, and which probably reach back to the beginning of the disease, while the posterior portion shows fresh otitic changes, which pass over to the base and cause bony ankylosis. Only the latter, which brought about a complete immobility of the stapes, could cause the high degree of deafness which was found before the death of the patient.

These observations are the more important, since, in spite of the ankylosis of the stapes, the patient heard the voice at 1 m. distance right side, and one half m. left, and on the right side heard whisper, also, at 2 cm. distance, and the possession of these remnants of hearing can well be explained by the fact that a large part of the membrane of the round window still functionated. This case is in sharp contrast to one described by me in a previous number of this journal* where there was found, with a complete occlusion of both round windows, good hearing on the side where the stapes was still movable.

CASE IV.

George Sch., 27 years old, was brought, July 2, 1893, unconscious to the nerve clinic, and died there the next day. In regard to his disease, there could be learned only that, one day before, he was seized with a spastic paresis of the left upper extremity, which became somewhat better in the evening. Nevertheless general convulsions and unconsciousness supervened, on which account he was brought to the hospital the next day. It was said, also, that he suffered with his right eye and left ear, and several days before had taken a large dose of potassium iodide on account of his eye trouble. Previously he had had a squint in his right eye, which condition had become worse in the last few weeks. I can pass over the findings on his entrance to the nerve clinic, as they had no direct connection with his ear trouble, and will add only that a test for hearing could not be made, but that ac-

*Archiv. für Ohrenkeilkunde, Bd. LIII. S. 61.

according to the belief of the physician who treated him, the patient could hear well.

The necropsy was held July 5, in the pathologico-anatomic institute and the pathologico-anatomic diagnosis was: Arteritis of the internal carotid. Progressive thrombosis of left internal carotid. Multiple encephalitis left hemisphere; post necrotic thrombotic cyst of the right hemisphere; meningitis dispersa, cerebral compression, lobular pneumonia.

As there was supposed to be an ear affection on the left side, Eppinger gave me the left temporal bone for further examination. On cutting the bone I found the pars petrosa comparatively small, the internal meatus abnormally wide, measuring at its internal end 9 mm. in breadth, and 8 mm. in height. The drum white and thickened, and showed a large perforation posterior inferiorly, which also reached into the posterior superior and posterior quadrants. The bone of the temporal bone was very sclerotic, the antrum mastoidum very small, and few cells were to be found in the mastoid, near the antrum. The mucous membrane of the middle ear appeared greatly thickened and swollen, and covered the windows and the large ossicles.

MICROSCOPIC FINDINGS.

INTERNAL EAR.—The dilatation of the internal ear extended outward as far as its end, and the usual thick bone between the ganglion canal and the internal meatus had almost disappeared, so that the internal meatus reached nearly to the ganglion canal of the middle turn of the cochlea. At the bottom of the meatus the measurements were 7 mm. Likewise the canal for the vestibular and facial nerves were very much widened, the latter to its entrance into the middle ear. The periosteal covering of the meatus was thickened and contained numerous spindle cells. The nerves were comparatively thin, and contained considerable blood in their sheathes. Between the bundles of the vestibular nerve, especially between those of the branches to the posterior ampulla, were numerous round cells and leucocytes. Signs of them were also found between the bundles of the cochlear and facial nerves. In the cochlea, considerable pigment was

seen, which was present in large amounts in the stria vascularis and the lamina spiralis of the middle and upper turns, and in smaller amounts in the endosteum and ligamentum spirale. Fresh hemorrhages were visible at several places on the lamina spiralis. The parts of the cochlea, especially the organ of Corti, are very well retained, though they appear in general somewhat more delicate and less well developed than usually. The nerve fibres in the end portion of the basal turn are evidently smaller, and there is more connective tissue here and in the ganglion canal around the nerves and ganglion. The aqueductus cochlearis, like the internal meatus, is very wide, especially its inner half, but its outer half plainly, also. There are no special pathologic changes in the vestibule and semicircular canals with the exception of somewhat numerous and profuse hemorrhages.

MIDDLE EAR.—The mucous membrane of the tuba Eustachii shows a great development of folds, and there are present numerous larger or smaller foci of inflammatory infiltration. Only a few sections of the drum are good for examination.

In these are seen a large involution of the border, a large cicatrix at the border of the membrane, and a severe inflammatory infiltration of the mucous membrane with separation of the layers. The lining of the middle ear is inflammatorily swollen and infiltrated to a high degree, along the entire inner wall. Only in the regions of the windows and on the inferior wall are the conditions somewhat different since here the deeper spaces are filled out with a loose connective tissue, and only the superficial layers of the mucous membrane, or only several broad or rounder foci are infiltrated with round cells. Similarly infiltrated are some cone like projections on the inferior wall. Most of the epithelium is retained and only at the places of greatest infiltration is it no longer to be seen. The niche of the oval window is filled out with loose connective tissue which is sprinkled with large swollen cells, occasional round cells and leucocytes. Between the bars of the stapes is a large cystic space. The bars are very narrow, stain deeply with hematoxylin, but their bony structure is no longer demonstrable. Very irregular conditions are found in the base of the stapes, whose bone has almost entirely disap-

peared, while the annular ligament shows numerous spindle cells, and in places is calcified. At the anterior, superior edge, the annular ligament borders directly on the diseased bone, and here the most internal portion of the anterior stapedial bar is bound fast to the bone.

The anterior portion of the round window is stenosed by a hyperostosis of the bony wall, and this hyperostotic bone has the same structure as the promontory. The remaining portion of the anterior half of the round window is filled out by thick connective tissue in which several cyst like spaces are enclosed. The posterior half of the niche is free, but its mucous membrane lining like that of the middle ear shows great inflammatory infiltration, is and likewise the membrana tympani secundaria on its external aspect. The sinus tympani shows only a small lumen, and otherwise is filled out with dense fibrous tissue, that in its superficial portion is somewhat infiltrated. The lining of the antrum mastoideum is similar to that of the middle ear.

THE BONE.—The pars petrosa is somewhat smaller and, as mentioned above, the canals connected with the cranium, viz.: internal meatus and aqueductus cochlearis, are enormously dilated, the latter nearly to its junction with the cochlea. The bone shows everywhere manifest sclerotic changes, very few cells, an intense staining with hematoxylin, and a scant staining with eosin, which allows the conclusion that there is an increased amount of calcareous matter. The mastoid shows only a few small cells below the antrum, and it, as well as the squama, as far as the latter was examined, is sclerotic with a few small cellular spaces filled with loose connective tissue and wide vessels.

The bone of the internal wall of the promontory is especially altered. Everywhere under the periosteal layer of the mucous membrane are short and wide canals in the bone, in which is only scant or no connective tissue. The superficial bone lying between stains unusually deeply with hematoxylin especially in the anterior part of the middle ear, up to the niches of the windows. In addition to these old changes in the bone, a fresh ostitic lesion at the same place as in the above described cases is found in the anterior circumference of the stapes. It extends from the oval window

into the promontory forward and ends here between the posterior border of the middle turn and the end of the basal turn of the cochlea. Outward, it reaches, for the most part, the periosteum, inward to the endosteal covering of the sacculus rotundus, while upward it is separated from the facial canal by a small, still normal layer of bone. Posteriorly it surrounds the fenestrum ovale, and ends at the inferior border even before the middle of the niche, while at the superior it stops just before the posterior border of the niche. This lesion resembles exactly the foci in the previous cases. The whole bone, as far as it is diseased, is traversed by large, partially round, partially more oval spaces, in which are very dilated vessels, granulation cells, in places large multinuclear osteoclasts and more often layers of osteoblasts along the walls. The place of the bone is taken by a simple osteoid tissue, in which, at isolated centrally lying places, are still to be seen long remnants of old bone. Stained with hematoxylin, they show an indistinct structure, without distinct bone corpuscles. In some of the spaces which contain vessels, we find, close to the vessels, some cells with deeply staining nuclei, like the lymphocytes. The diseased bone at the border of the foci is sharply defined from the healthy, and it seems that a part of the wall of the vessel space borders directly on the healthy bone. Here the bone corpuscles at places in the healthy bone show themselves altered in so far that their lumen is somewhat widened and the bone cells in them are plainly visible. This gives the impression that the process consists not only of a resorption of the old bone by osteoclasts and formation of new by osteoblasts, but also of a reconstruction of the old bone, in which the cells of the old bone at the same time play an active part. It must still be said in regard to the entire lesion, that the disease in its entire extent is comparatively fresh, and that nowhere are sclerotic portions nor such as show canals filled with medullary or old connective tissue. The most numerous, and therefore the freshest, are in the disease portion at the anterior periphery of the lesion, and at the posterior part, in the circumference of the stapes, where a part of the stapes and the annular ligament is affected by it, so that a bony ankylosis could have been expected if the disease had lasted longer.

In this case, pathologic changes of different durations and kinds were found in the temporal bone. The abnormal smallness of the temporal bone and its extremely sclerotic bone, together with extreme dilatation of its internal meatus and a large part of its Fallopian canal presumably was caused in the early period of patient's life, and the patient at this time probably suffered from hydrocephalus and rachitis. When the purulent otitis media appeared, which caused the perforation of the drum and the changes in the mucous membrane and superficial bony layers of the internal wall of the middle ear, cannot now be told, as the history is silent on this point. Finally there is the third change, viz.: the fresh otitic lesion in the neighborhood of the oval window, in regard to whose origin several theories could be advanced. To consider it as the sequel of the otitis media purulenta does not seem correct according to the other findings, and when I demonstrated the specimen at the 7th German Otologic Congress at Würzburg, I advanced the theory that it was a disease of the bone due to syphilis, since definite signs of syphilis were found both from the history and from the necropsy.

I can refer to a case of Schwabach* at Würzburg in which similar signs of syphilis were present, although Schwabach himself did not seem to give this construction to his case.

CASE V.

Ch. Magdalena, 34 years old, workingmen's wife, from Graz, was brought to the obstetric clinic, January 4, 1896. Labor had commenced 48 hours before, and the patient had been examined several times by the midwife. She was suffering with fever (38.2°), there was a cross presentation, prolapse of the umbilical cord, and living child. After podalic version the patient was free from fever for four days, when the typical symptoms of puerperal septicemia set in, from which the patient died on the 8th day of the disease, on the 11th day after delivery. The patient was very deaf, but could not tell exactly when the deafness first began. During preceding

*Zeitschr. f. Ohrenheilk., XXXI., P. 122.

pregnancies (I 1893, II 1894) the deafness increased. Tests for hearing were not made, and I could establish only that one had to shout to her before she could understand. Her husband, whom I subsequently questioned about the ear trouble of his wife, was suspicious and evaded every inquiry; he insisted he knew nothing of his wife's aural trouble.

The necropsy on January 15, 1896, in the pathologic anatomic institute, gave body small, weak, abdominal wall inflated. Brain substance soft, delicate, rather rich in blood, ventricles fairly wide, cerebellum similar to cerebrum. Subcutaneous tissue without fat. Heart small, cavities narrow, musculature brown, fragile, valves correspondingly formed. Left lung large. Pleura delicate, upper lobe contains air, pale and streaming with frothy liquid. Lower lobe sprinkled by confluent hepatised foci. Bronchi reddened, covered with tenacious mucous. Right lung likewise large, much fibrinopurulent exudate in the pleural sac. Tissue of upper lobe contains air, edematous; the lower lobe atelectatic. Throat organs show nothing abnormal. Peritoneum covered with a mass of pus as are the loops of the gut, which are adherent to one another. Spleen large, capsule delicate, tissue greyish brown, pulpaceous. Both kidneys large, very brittle, pale and fatty colored. In stomach large amount of contents, mucous membrane in longitudinal folds, reddish grey colored. Scanty contents in intestinal canal. Mucous membrane in places reddened and swollen. Liver large, very heavy, brittle, yellowish colored. In the bladder, a little cloudy urine, mucous membrane somewhat reddened. Uterus very large. Inner surface covered with dirty, foul mass. Placental attachment necrotic. Tissue of the uterus friable, the lymph vessels filled with pus. Ovaries greatly increased in size, tissue moist and reflecting, grey. In them, are several foci, size of lentil, containing thickened pus.

DIAGNOSIS.—Peritonitis purulenta, pneumonia lobular; atelectasis pulm. dextr; pleuritis dextr; metrolymphangit; endometritis; ophoritis ascendens; sepsis puerperalis.

In the right temporal bone, which I obtained for examination, I found the epidermis in the external meatus detached in a large lump (the specimen had been lying in Müller's fluid); in the middle ear and antrum, mucous secretion. The

bone, on sawing, was very hard, the antrum comparatively small, and only a few pneumatic cells were to be found behind it. While separating the squamous portion from the petrous, the drum was unfortunately torn, and the largest part remained on the inner portion, while only the anterior, smaller part on the outer. The mucous membrane of the ossicles, the antrum and the middle ear, as far as could be seen, seemed to be greatly thickened. The recognition of the condition of the mucous membrane was rendered difficult by the action of the Müller's fluid.

After decalcification of the specimen in nitric acid, the microscopic examination was made.

MICROSCOPIC EXAMINATION.

INTERNAL MEATUS.—Of the nerves, the greater part of the right cochlear and a large part of the vestibular was wanting in the specimen. In the remaining parts, there were considerable venous hyperemia and large hemorrhages between the external nerve sheathes, and small punctate between the nerve bundles.

COCHLEA high degree of venous hyperemia, and a considerable amount of pigment in the periosteum of the scala vestibuli. In the reverse portion of the basal turn, a large stretch of the ganglion spiral was wanting as well as the nerves belonging to it and the corresponding organs of Corti, while the stria vascularis, promentia spiralis and Reissner's membrane were retained. In the lamina spiralis there were only pigment, scant connective tissue and a few fat droplets, in Rosenthal's canal considerable connective tissue at one place, in the remainder only scant connective tissue and some fat droplets, Corti's organ failed for a wider stretch than the spiral ganglion, as it failed even where a few ganglion cells were visible. In the lowest part of the cochlea, the organs of Corti were well preserved, but even here in the ganglion canal, a lessening of the nerve cells and an increase in the connective tissue were to be observed.

Nothing pathologic was to be seen in vestibule and semicircular canals.

EUSTACHIAN TUBE. The mucous membrane of the cartila-

ginous tube is rather thickened, and in places there is a more intense accumulation of round cells and in places numerous formative and spindle cells.

The middle ear is for the most part filled out with dense connective tissue with a few spindle cells, and a richer, homogenous intercellular substance, and this connective tissue is connected with the lining of the internal wall, which likewise is extremely thickened, but which is entirely distinct in structure. It contains numerous vessels and cells, especially spindle cells, and some long cyst-like spaces, lined with cylindrical epithelium, which are larger and more numerous in the connective tissue which fills out the niches of the windows. They are filled filled with mucous and degenerated cells. Still larger and more numerous are the cysts along the inner wall, and they are often so arranged that a large number lie side by side, separated from one another only by a thin wall. The space in the posterior half of the middle ear has disappeared except for the spaces mentioned. The anterior half is somewhat different. The middle of the drum is adherent to the internal wall and so shuts off the anterior, inferior portion of the middle ear, in which, however, the mucous membrane is likewise thickened by hypertrophy of the connective tissue, and there is also found in it numerous, cyst-like spaces, round on cross section, which permeate the entire mucous membrane especially in the inferior internal wall of the middle ear. The drum shows a cicatrix in the superior anterior quadrant; the substantia propria is lacking and the epidermis borders directly on the connective tissue that fills the middle ear.

As to the chain of ossicles, the long process of the anvil and the stapes are surrounded by connective tissue, and the manubrium of the hammer is bound to the promontory by connective tissue. In the joints between the hammer and anvil, and between anvil and stapes, the articular cartilage is calcified, especially on the stapes. Large masses of chalk are deposited in some of the medullary spaces of the body of the anvil. The posterior bar of the stapes is retained, but is adherent by means of connective tissue to the posterior wall of the niche; only remnants of the anterior remain.

All of the inner two thirds is destroyed except a few calcareous remnants in which no bone corpuscles are to be found, and these are surrounded by dense connective tissue, and entirely enclosed in an exostosis having its origin in the niche.

The antrum is divided by connective tissue septa into a number of small spaces, all of which are filled with mucous or mucous swollen epithelium. The mucous membrane lining of the antrum as well as that, of the surrounding and even distant pneumatic cells is extremely thickened by new-formed connective tissue, and in some of the cells of the neighboring bone are osteoblasts and clear, eosin-staining areas of new-formed bone.

The bone was pathologically altered in several ways. In the first place, there was an unusually high position of the bulbus jugularis, and thereby the bony floor of the middle ear was reduced to a thin plate, which at places showed large holes in the bone. Furthermore, the bulbus had caused the disappearance of a part of the pars petrosa, and reached to the under border of the promontory, posteriorly to the ampulla of the posterior semicircular canal, which was separated from the bulbus by a layer of bone as thin as a sheet of paper. Then, there was a somewhat large hyperostosis of the promontory which began just at the annular ligament of the stages and gradually increasing in circumference reached to the inferior border. Here excrescences (osteophytes) projected still further downward, which partially obstructed the passage to the niche of the round window from without and above. The new formed bone was periosteal bone, and showed in the middle, at about the height of the round window, a hole that was filled with a fibrous connective tissue rich in cells. Internally these hyperostoses as well as a similar one on the under wall, narrowed the niche of round window very much, and the small remnant of the window, as already described, was filled with connective tissue. Such a stenosis of the niche of the window by hyperostoses of the bone is not seldom seen, and has been described by me before. The new-formed bone is scarcely distinguishable in its structure from the other periosteal bone of the labyrinth capsule, only staining somewhat deeper with eosin.

Decided disease of the bone was found in the niche of the oval window. In connection with a chronic inflammation lesion in the bony, there are seen here, on the surface, several large broadbased exostoses which have developed as follows: one outward and downward from the inferior border of the base of the stapes, the second, on the same side above the stapes, and the third from inferior, external border of the bony facial canal. The lesion in the bone corresponds to the anterior end of the base of the stapes, and below has a breadth of 4-5 mm. surrounds the base of the stapes at the anterior third, and extends from the middle ear to the periosteal lining of the vestibule, or the end of the basal turn of the cochlea.

Only the anterior part of the base of the stapes is affected with bony ankylosis; elsewhere the joint is free and the exostoses are found only in the niche above and below, and they posteriorly become smaller and smaller and lie even further outward.

The diseased bones show greatly dilated vessel channels, traversed by wide vessels, in whose vicinity are numerous formative cells, in some places large multinuclear giant cells, osteoclasts, and in places also osteoblasts. The bone nearest to the canals consists often only of osteoid tissue, which, especially in the middle of the lesion has entirely replaced the vanished bone. The diseased bone is everywhere sharply differentiated from the healthy, and at the promontory a few dilated channels run downward, which are bordered at their peripheries by layers of osteoblasts and osteoid areas. At the superior border of the focus are irregular round protrusions of the diseased into the healthy bone and at the anterior border of the focus, at the posterior periphery of the superior turn of the cochlea are numerous, irregular masses of calcium salts in place of the old bone.

The bone in the vicinity of the drum and meatus was little altered; at the periphery of the bony tube, and at the posterior inferior part of the pars petrosa, it was very osteoparetic with wide medullary spaces. In the posterior part of the pars squamosa, toward the antrum, and at the superior wall of the middle ear, were large spaces in the bone, filled out with a cellular infiltrated, inflammatory connec-

tive tissue. At their peripheries were beautiful osteoblasts and new formed layers of bone.

Since nothing certain about the ear affection of the patient could be learned from the anamnesis, we must make our judgment from the anatomic findings alone. According to that, it was a case of long continued inflammation of the middle ear, which caused a perforation and later a cicatrix of the drum, filling the middle ear and part of the antrum with connective tissue, and in places lesion of the superficial bone. Worthy of note was the larger focus of inflammation in the bone of the anterior periphery of the oval window, which, starting here, reached to the wall of the vestibule and the basal cochlea turn, and caused bony ankylosis in the anterior third of the base of the stapes. This focus showed, in the periphery of the oval window, comparatively old ostitic changes, which became fresher the more anteriorly they were, so there can scarcely be any doubt that it took its origin from the anterior portion of the niche of the oval window, and progressed from here gradually forward to the vicinity of the vestibule. As a whole, however, the ostitic lesion cannot have lasted a long time, since all the changes are of a comparatively young kind.

Of special note are the filling out of the middle ear* and niches of the windows with connective tissue, and the numerous, cystic spaces in the middle ear and antrum, which, lined with cylindrical epithelium, are to be regarded as remnants of the original middle ear—mastoid cavity, furthermore the extreme thickness of the floor of the middle-ear, and the bone of the inferior wall of the posterior semicircular canal caused by the high position of the bulbus jugularis, and, finally, the changes in the end portion of the basal cochlear turn. The explanation of the disappearance of the ganglion cells, and the increase of connective tissue in Rosenthal's canal could best be explained by the supposition, that the

*An obliteration of the middle ear by connective tissue which von Tröltzsch has already described (these archives Bd. VI, p. 73) has been observed by me and portrayed in Schwartz's Handbook Bd. I, p. 253, Fig. 18, so that Holzel's statement (Zeitsch. f. Ohrenheilk., Bd. XLIII, p. 176) that there is nothing about it in the literature is incorrect.

widespread inflammation in the middle ear had also attacked the end portion of the basal cochlear turn.

CASE VI.

Marie M. 76 yr. old widow, was examined at the medical clinic where she was being treated for carcinoma of the stomach. She said she had suffered from deafness for 20 years, yet could tell nothing definite about its cause. Nothing could be found out about other ear symptoms from her as she could neither read nor write.

The drums, which consisted of white scars, were greatly retracted, with extensive cloudiness and bending of the borders. The left drum was the whiter. The mucous membrane of the nose was pale red, the right lower turbinate somewhat atrophic, and there was a large amount of mucous secretion. The posterior pharyngeal wall showed numerous granulations, and considerable mucous was also found in the naso-pharynx. Test for hearing gave a negative result in both ears with every method, watch, voice, all the tuning forks with the single exception, that on the left side, the deep tuning forks c^1 , c^3 were still heard by bone conduction. The deep and large tuning forks C, C_1 , C_2 , were unfortunately not tested for bone conduction, but could not be heard by air.

The patient died as a result of her stomach cancer, February 2, 1896, and the necropsy was made February 3, in the anatomico-pathologic institute.

Post-mortem findings of February 3, 1896. Body small, slight frame, emaciated, abdomen greatly distended, greenish discolored. Cranium large, roundish oval, compact. Meninges rich in blood, pia greatly thickened, brain substance friable, soft, cortex atrophic. Medullary substance permeated by points of blood which were easily wiped off. Ventricle dilated, filled with clear serum. Cerebellum more friable than cerebrum, vessels at the base very rigid. Pons and medulla strong and tenacious. In the basal sinus was dark, clotted blood. Subcutaneous areolar tissue devoid of fat. Heart small, inclined, contracted, cavities narrow, muscle friable. Valves correspondingly formed. Both

lungs large, light, pleura delicate, upper lobes contain air, pale and emphysematous at the borders, on section covered with a frothy liquid, the lower lobes containing numerous hepatic foci. In the bronchi tenacious mucous, mucous membrane greatly injected. Mucous membrane of pharynx of livid color, that of the esophagus and trachea pale and smooth. Thyroid gland enlarged. Spleen small, tissue red brown, pulp scanty. Both kidneys small, surface smooth, tissue red brown, firm, tough. Stomach very wide, filled with fluid products of digestion, mucous membrane in fundus ecchymotic. smooth, the pyloric region is occupied by and its tissue incorporated in a tumor of about size of fist, which consists of soft, pale gelatinous tissue and so narrows at the pylorus, that it scarcely allows the little finger to pass through it. The large omentum is studded with larger or smaller nodules of gelatinous tissue, as is the under surface of the diaphragm. The other organs show nothing abnormal.

Diagnosis, carcinoma gelatinos, partis pylorici cum metast. in oment. maj. et diaphragma. Marasmus.

The temporal bone which was given to me by Eppinger, for examination, showed the following findings.

RIGHT EAR.—The mucous membrane of the tube and anterior and inferior half of the drum thickened. The hammer-anvil articulation movable. The niche of the round window entirely overgrown with tissue, the lower part of the round window also. The bars of the stapes are very thin. The mastoid process very pneumatic, and the pneumatic cells reach for backward beyond the area of the sulcus sigmoideus. The bulbus jugularis is also very extensive.

LEFT EAR.—The drum thickened, greyish white, somewhat transparent at the anvil. The mucous membrane of the middle ear thickened on the inner wall, the niche of the round window for the most part overgrown, that of the oval narrowed from below, and the anterior bar of the stirrup ankylosed with the inferior border of the thickened wall of the niche. The bars of the stirrup are very thin, and the niche above the bar is free. The mastoid process very pneumatic, and apparently not pathologically altered.

The specimens were, as usual, after fixing in Müller's fluid, decalcified in 5 per cent. nitric acid solution and examined microscopically.

MICROSCOPIC FINDING.

RIGHT EAR.—INTERNAL EAR.—In the specimen, only remnants of the nerves in the internal meatus are to be found; the remainder was torn off in removing the brain from the cranial cavity. With the exception of hemorrhage into the sheathes these remnants show no pathologic changes.

In the cochlea, the nerves and ganglion cells in the superior turns are well retained; in the basal, the number is greatly lessened, and the vicinity of the ganglion canal is greatly calcified, and the connective tissue increased in the canal itself. Both sheaths of the spiral lamella of the basal turn are changed into masses of chalk, in which no bone structure can be made out, and the space between them is very much narrowed. The organs of Corti show no unusual changes; only in the basal turn are the cells less well retained. The periosteal covering of the scalae and the external portion of the ligamentum spirale, where they border on the diseased bone, are more or less greatly thickened, and the latter especially in the superior turns, is changed at places into a thick layer of connective tissue deeply staining with eosin. In the vicinity of the venous channels in the basal turn, as well as in the cellular spaces in the cochlear spindle, the bone contains large amounts of chalk, and similar changes extend upward into the dividing wall of the inferior and middle turn. The aqueductus cochlearis is stenosed at its external part, where it empties into the cochlea, and further inward entirely closed. Within the diseased bone remains of connective tissue and some particles of calcium phosphate represent the former canal. The medial end of the canal is retained. At the inferior part of the scala tympani, the diseased bone projects into the cochlea in the form of a rounded exostosis, and here the basal membrane is detached at a circumscribed place from the spiral band.

No pathologic changes are to be found in the vestibule and semicircular canals in spite of the extreme changes in

their bony walls. The walls of the semicircular canals show some small prominences of diseased bone which project into the lumen; the endosteum is in places somewhat richer in cells and thickened.

MIDDLE EAR.—The lining of the bony tube (the cartilaginous fails on the specimen) shows a sclerosis in the superficial layers. In the tympanic cavity the lining consists of fibrous connective tissue with considerable intercellular substance and few nuclei, and only in spots is an infiltration of spindle cells. In places this thickening of the lining attains a higher degree, especially in the niches, which are closed by fibrous connective tissue where they are not already narrowed by diseased bone. Only in the anterior superior part of the niche of the oval window does there remain a part of the lumen, in spite of the thickening of the lining and connective tissue adhesions.

The drum, as a whole is somewhat thickened; especially at the anterior border, and its mucous membrane layer is formed similarly to the lining of the tympanic cavity. The dermal layers consists of a thin layer of fibrous connective tissue, while the substantia propria shows no change. A special thickening of the lining is found immediately over the drum at the external side of the attic. The bony wall here is uneven, with excavations, with sharp projecting spiculae, between which the thickened lining dips in.

Of the ossicles, hammer and anvil show similar changes to the neighboring bone of the external wall of the middle ear, dilated medullary spaces and vessel channels, new formation of bone, and deposit of chalk in the articulation, the latter also being present in the anvil-stirrup articulation. The stapes has very slender bars externally; inwards, the anterior is firmly bound to the wall of the niche, the posterior partially so. The latter shows furthermore a superimposition of a thick layer of greatly calcified bone. The anterior part of the base is thickened from without inward, by a hyperostosis similar to that of the posterior bar. Internally, the bone shows signs of chronic inflammation, filling out of medullary spaces present with the connective tissue. The posterior part of the base is firmly attached to the surrounding bone (bony-ankylosis); nothing can be found of the annular ligament.

and the same pathological changes are found as in the bone of the petrous portion. The anterior part of the base of the stapes is dislocated out of its articulation outward, for about the thickness of its base, and there bound by bony adhesions to the diseased bony wall. Its bars and head are bent downward, enclosed in fibrous connective tissue and adhering to the wall. The larger vessels along the tenso tympani and nervus petrosus shows clearly thickening of the intima through endarteritis.

The mucous membrane of the mastoid process shows a looser structure than that of the middle ear, and, as far as the disease of the bone reaches, is denser and contains numerous spindle cells. Some small cells are entirely filled out with connective tissue.

BONE.—The bone was diseased for a great extent, and a part still is. In general it can be said that so far as it borders on the tympanic cavity, it shows pathologic changes of greater or less breadth, and that the greater part of the bone surrounding the semicircular canals is diseased. In the anterior part of the tympanic cavity, the disease of the bone reaches about 4 mm., into the bone of the internal wall, attacks certain turns of the cochlea and extends to the anterior periphery of the internal meatus, which is involved for a large extent in the process. Further posteriorly, the internal border is found at the periphery of the sacculus and utricular, where the points of nerve entrance are likewise partially surrounded by diseased bone. The deepest point of the disease is the region of the semicircular canals where it has attacked the bone in the region of the aqueductus vestibuli and also a part of the vicinity of the semicircular canals. Furthermore, the entire bony floor of the middle ear, a large part of the bony capsule of the bulbus jugularis, as well as the vicinity of the bony tube and Haller's space, and the parts of the external meatus adjoining the middle ear are pathologically affected in a similar way. I can make no statement about the superior wall of the middle ear or the external part of the mastoid process, as these were not examined histologically.

In spite of this apparently diffuse process, with careful examination, there can be found the same foci in the bone as in

the other cases, only on account of their great extent they have fused with one another. The lesion in the internal meatus shows the bone ulcerated for a great distance, and a little fibrous connective tissue lies in the cavity, whose floor seems irregularly bitten out. The adjoining portion of bone show somewhat larger vessel channels, and everywhere sclerosis with deposit of calcium salts. There is a new focus which begins broad at the inner wall of the aditus, and surrounds the external semicircular canal, from without inward, and a second, deeper one, in the region of the sinus tympani, which internally surrounds the posterior semicircular canals and affects the entire bone of this part up to the external wall of the aqueductus vestibularis and comes to an end only at the upper periphery of the petrous pyramid. In this most superior part, the disease appears the most acute. Here the disease of the whole floor of the tympanic cavity is evident. The bone here shows a deep bluish-yellow stain (hematoxylin eosin) and is apparently, as a whole, dense, with hypertrophy of the connective tissue, in the cellular spaces, as in the medullary spaces, layers of osteoblasts on the bright red internal bony wall are found. Close to the older bone, with plain lamellar formation and dark blue staining, is bluish red bone, with less distinct lamellation, and greater prominence of the bone corpuscles, and superficially, around the bony borders, faintly staining layers with rows of osteoblasts,

LEFT EAR.—In the internal meatus, there are also hemorrhages between the nerves, and the periosteal covering, where it borders on the diseased bone, is thickened and permeated by numerous spindle cells. In the cochlea, the nervous apparatus is well retained, and the endosteum of the end portion of the basal turn greatly calcified. The diseased bone projects in the form of an exostosis into the lumen of the scala tympani, and the apical turn. The aqueductus cochlearis is partially interrupted by fibrous, calcified connective tissue. From the apex of the cochlea, and from its sides, the disease of the bone has advanced into the spiral of the cochlea, and furthermore, the bone in the neighborhood of the medullary spaces of the cochlear spiral is very calcified. The ligamentum spirale bordering on the diseased bone, is, as in the right ear, greatly thickened.

The same conditions in vestibule and semicircular canals as on the right side; an alteration in the part of the external semicircular canal bordering on the antrum is noteworthy, as it has a more acute form, owing to the reaching in of the diseased bony wall.

MIDDLE EAR.—The mucous membrane lining is similarly constructed to that of right. The niches differ somewhat. The lining of the niche of the oval window is markedly thickened, but the lumen is otherwise rather free. The posterior bar of the stapes, whose bone is greatly calcified and no longer shows bony structure, adheres, to a large extent, to the niche's wall by fibrous connective tissue, while the anterior bar is displaced somewhat outwardly with the corresponding part of the base, and here likewise appears firmly bound to the wall of the niche. The cartilaginous layer of the base of the stapes is retained for the larger part of the periphery, although partially calcified, and the greater part of the annular ligament has the same appearance, but the cartilaginous layer of the wall of the niche is for the most part lost, while the diseased bone reaches more or less into the base of the stapes.

In the posterior superior part there is a complete bony ankylosis of the base with the pars petrosa. The base, in its middle and posterior part is very thin, consisting especially of the cartilaginous layer internally to which as the thin calcified connective tissue layer, and externally a thin layer of calcified structureless tissue.

The anterior part of the niche of the round window is somewhat stenosed by exostoses of the diseased bone, and the lumen is completely filled out by fibrous connective tissue in which remain small spaces lined with epithelium.

In the most anterior internal part, this tissue is somewhat less dense and shows wider spaces in the network. The posterior part of the niche is completely closed by bone. Hammer and anvil in this ear scarcely altered.

The bone on the left side, in the region of the tympanic cavity is diseased similarly to the right. Around the tube, the bone is extremely osteoporotic, with numerous medullary spaces and large vessel channels which show at their periphery spaces of osteoid tissue. Spaces entirely filled out with

connective tissue are found here with borders often jagged and irregular.

Beneath the cochlea and vestibule are large pneumatic spaces, whose lining of mucous membrane is thickened and impregnated with numerous spindle cells and in whose vicinity, as in the entire periphery of the middle ear, the bone shows itself diseased into its depths, as in the right side. In aditus and antrum are also found some circumscribed otitic lesions on the superficies of the bone, and likewise deeper ones in the region of the superior and posterior semicircular canals, which are apparently isolated, but in reality the upper are in communication with the diseased medullary spaces of the upper face of the pyramid, while those around the posterior semicircular canal are joined with the especially well developed cellular spaces in the inferior part of the posterior face of the pyramid.

In the internal meatus is a large hole in the bony wall, somewhat larger than on the right side, with very uneven jagged floor, and the periosteal lining over it is destroyed. On the floor of the hole, some fibrous connective tissue lies on the greatly diseased bone, which is traversed by large vessel channels filled out with fibrous connective tissues, rich in spindle cells, and surrounded by a completely sclerosed bone. In some larger vessels, e. g., the artery of the fossa subarcuata there is an evident thickening of the intima by endarteritis.

REVIEW.—The most important feature of this case, which distinguishes it from those previously examined, is the extreme extent to which the bone of the entire surrounding of the middle ear is involved although the disease has not everywhere penetrated to the same depth in the bone, starting from the middle ear. Furthermore, it is noteworthy that not only the same appearance of the diseased bone, but also another circumstance argues for a similar disease of the bone in this and in the preceding cases. An exact examination of the specimens shows that the disease took its origin from the same places in this as in the other cases, and thence spread, only here the foci have fused on account of their greater extent, and the other walls of the middle ear, the antrum and the tube were similarly diseased. The dis-

ease of the bone in this case, also showed at different places an altered histologic picture. Thus, especially at the borders, completely sclerotic parts were found near others in which the bone showed very dilated vessel channels, which, after Siebenmann, can be called spongy. Near those parts in which the disease had years before come to an end, places were visible, where there were plain signs of an acute progressing disease. The distribution of the freshly diseased parts and the old retrograde changes agreed in the main with the theory that I advanced in the former cases. The picture on the under wall of the middle ear and the vicinity of the bulbus jugularis is somewhat different from that of the other petrous portions, and here resembles rather that which we observe in the purulent middle ear inflammation. These differences can possibly be due to the different structure of this bone and less to another cause of disease.

As to the duration of the trouble, we can assume from the statement of the patient that she was deaf for 20 years, that the disease of the bone which we must regard as the cause of the deafness, on account of the change at the windows, lasted at least that long if not longer.

Of the changes, special note must be taken of the luxation of the stapes outward which was caused by the inflammation destroying the connection of the base of the stapes with the bone, or changing it into a loose new connective tissue, so that the stapes obeying the pull of the muscle, was directed outward and then became firmly bound to the wall of the niche.

The complete deafness of the patient in the right ear, and nearly complete in the left finds its explanation not only in comparatively insignificant changes in the labyrinth, but, as I have pointed out in other cases, can be referred to the closure of the niches of the window and the fixation of the stapes. What influence on the hearing the bilateral occlusion of the aqueducti cochleares had cannot be determined on account of the large number of weighty lesions which were present. It seems that the communication between perilymph and sub-arachnoidal space through the internal meatus can allow a partial occlusion of the aqueductus cochlearis. Why, in the left ear, the deep tuning forks c^1 , c^2 , c^3 , (unfortunately no

test was made with the deeper ones) were still heard by the bone conduction from the mastoid, can be explained in my opinion by the fact that on the left side, not only was a large part of the annular ligament of the stapes retained, but also because the base of the stapes for a considerable space consisted not of bone but of a thin cartilaginous layer, over which, externally, the lining of the middle ear lay.

Whether the patient in earlier life suffered from dizziness could not be found out, as she was deaf and could not read, but it is not improbable that she had attacks of dizziness like the other earlier case where the disease of the bone reached to and involved the passage of the ramus vestibularis.

CASE VII.

Marie B., 28 years old, property owner's daughter, suffered from infancy with epilepsy, and intense excitability, especially at the time of menstruation. In her home certificate she is designated as an idiot. She spoke few words, but the doctor who treated her in the hospital said her hearing was good. Three weeks before she had given birth to a healthy child and died in an epileptic attack, with which in the last weeks she had often suffered, and as a result of which, before her death she was unconscious for days.

Post-mortem April 13, 1899, in Prof. Eppinger's-pathological-anatom. institute.

Body small, rachitic build, thin, Skin earth-colored, lower portion of the abdomen wrinkled, covered with striated grooves; nipple region and linea alba very pigmented; pericranium sprinkled with some very circumscribed, fresh, macular hemorrhages. Pia of the base of the brain congested to the highest degree, the vessel walls delicate. Cranium rather small, long oval, thick at the base (12 mm.) everywhere clearly compact. The dura adhered firmly, somewhat thicker, and congested. In the superior transverse sinus, fluid, dark blood. The pia of the convexity little thickened, stretched, in general injected, the pial veins, especially the left side, clearly full. Brain weighs 1080 g.

The substance of the brain is of a hard friable consistence, and of a strikingly moist appearance. Cortex is hypertro-

phic dark greyish violet colored. Ventricles anteriorly a little wider; in the middle and posteriorly narrow. Fluid dark blood in the basal sinus.

Subcutaneous tissue rich in fat. Musculature thick and very dark. Dark fluid blood in the jugular.

Both lungs fixed, rather dilated. Remnants of the thymus as a bilobular loosely granular hyperemic organ, reaching to the base of the pericardium. A little clear fluid in the pericardium. Heart oblique, correspondingly large, on the apex a superficial milk spot. In the right cavities, fibrin; left, fibrin and clotted blood. Musculature of left heart a little thicker. The musculature of both sides is cross-striated streaked with light and dark, homogeneous on section, the valves very delicate and correctly formed.

Left lung small, rather heavy, everywhere containing air clearly rich in blood, and moderately edemic. In the middle of the posterior part of the lower lobe it is harder, friable, homogeneously granulated on section, dark reddish brown color, and containing fluid. Similar fluid found in bronchi. Mucous membrane intensely dark colored. Right lung, somewhat smaller, lighter, contains air, moderately edemic, tenacious mucous in the bronchi, mucous membrane of the pharynx dark colored, that of the esophagus stained with bile. Larynx and trachea pale. Thyroid gland rather large, aorta thin-walled, measuring 3.9 cm. in circumference above the diaphragm.

Position of abdominal contents correct, peritoneum rather rich in fat, spleen somewhat larger, hard, friable, dark-brown color, pulp large amount.

Left suprarenal normal; left kidney rather large, capsule delicate, easily detachable, surface smooth. Right kidney somewhat paler, otherwise like the left. Suprarenal capsule like the left.

The stomach contains bilestained fluid, mucous membrane normal thickness, slightly thrown into folds, bile stained. In intestinal canal, usual contents, walls medium thickness, mucous membrane here and there violet spotted. That of the small intestine, thin, smooth, homogeneously light violet colored. Pancreas of normal structure.

Liver of medium size, tissue hard, friable, dark brown color,

lightly yellow spotted. In the gall bladder, dark, blackish fluid bile.

In the urinary bladder, cloudy, pale yellow urine, mucous membrane normal. Vagina of proper length and width, mucous membrane hard, dark bluish violet in color. Uterus larger, puerperal malacious. Remnants of placenta in form of a plaque 1 cm. high attached to the anterior wall. Ovaries large and rich in follicles, without a true corpus luteum. Tubes of both sides free. Mucous membrane of the rectum very dark red.

Diagnosis. Hyperemia cerebi et meningium. Pneumonia sinistra. Uterus p. partum.

The examination of the ears made by me, showed the drums thick and of whitish color.

In the right tympanic cavity were connective tissue adhesions around the stapes, from the base to the neck, and the niche of the round window seemed to be completely occupied by connective tissue. From the middle of the promontory a splinter of bone projected downward, and another antero-inferiorly from it.

The left ear was similarly constructed, and here also were found adhesions around the stapes, and the niche of the round window occupied by connective tissue.

MICROSCOPIC FINDINGS.

RIGHT EAR.—On the floor of the internal meatus, profuse hemorrhage between the nerve fibres. In the acousticus, some small foci of altered nerve fibres. They are deeply and diffusely stained, their contour indistinct or not at all distinguishable, no corpora amylacea. As all reaction signs are absent, it is probably a case of post mortem change.

The soft parts of the cochlea in the upper turns have suffered somewhat through decomposition, while the lower are well retained. There are no pathologic changes.

The upper part of the posterior semicircular canal and the external portion of the external show the bony wall irregularly excavated.

Of the middle ear, the cartilaginous part of the Eustachian tube, whose lowest part is wanting in the specimen, shows a

lining consisting of fibrous connective tissue between whose upper layers a few long-oval and spindle shaped nuclei are found, while glands are entirely absent. The lining of the bony tube shows a similar construction.

The mucous membrane in the middle ear is very different in appearance at different places. While at places on the promontory it is so thin that, between epithelium and bone, a submucous and periosteal layer can no longer be found; at the superior part of the internal wall it is of almost normal appearance, and at the inferior part of the internal wall it is rather swollen, inflammatorily infiltrated, its vessels greatly dilated, the epithelium well retained, and a mass lies above an inflamed place on the surface, which consists near the surface of parallel, somewhat higher of a reticulated mass of fibrin threads infiltrated with pus cells.

The mucous membrane of the drum and ossicles is rather thickened and sclerotic especially near the border. Of the two large ossicles, the hammer is very sclerotic, as is the anterior part and the long process of the anvil, and the vessel channels show very dilated and filled vessels and an irregularly excavated surface.

In the niches is a similar fibrinous inflammation of the mucous membrane and a rich fibrino-purulent exudate lies on the mucous membrane, especially in the niche of the round window and the underlying, adjacent, excavated and cellular spaces of the bone. In addition, the niche of the round window is closed off by a broad connective tissue membrane from the middle ear, in its external portion. In the niche of the oval window are likewise thin, firm connective tissue adhesions between the bars of the stapes and the inferior wall of the niche, the promontory, and the facial canal above the niche. The stapes itself shows on its base anteriorly and externally, a medium-sized hyperostosis.

The mucous membrane of the aditus and mastoid process is not observably altered, yet some of the neighboring cellular spaces of the bone are filled with connective tissue, and bordered on their inner walls by small layers of osteoblasts and a red-stained area of bone. The bone of the pars petrosa shows a medium-sized hyperostosis, corresponding to the inner wall of the middle ear which extends posteriorly to

the windows, and is plainly demonstrated by its staining more deeply with hematoxylin. Fresher inflammatory changes are found in the pneumatic cells bordering on the middle ear, as well above as below the actual labyrinthal capsule. Their mucous lining is altered exactly like that in the adjacent part of the middle ear, and in the lower cells is the same fibrino-purulent deposit.

Some smaller cellular spaces show no lumen at all, but are filled out with fresh connective tissue with dilated vessels while osteoblasts are found on their bony wall. Beneath the diseased mucous membrane, the vessel channels in the bone are widened, as are the enclosed vessels, near to which are numerous developmental cells. The surrounding area shows likewise osteoblasts and new-formed area of bone staining very deeply with eosin.

A large lesion in the bone is found only in the external wall of the niche of the round window. The bone here, as a result of the fibrinous inflammation of the mucous membrane, although separated from it by the thickened periostum, is diseased along the entire wall, and the disease reaches beyond the region of the round window within the bone, further back toward the mastoid. The diseased bone is differentiated both by its structure and by different reaction toward the stains employed from the healthy. It does not, however, show the same appearance everywhere. In its most anterior part, are moderately dilated vessel channels, with few cellular contents, surrounded by a very calcareous bone, staining deeply a dark blue with hematoxylin-eosin, while in the posterior, larger portion, the lesion shows more reddish blue color. The bone here is everywhere traversed by very wide vessel channels in which are dilated vessels and numerous large cells. The cells of the bone show here a rounder form, indistinct nuclei, and a clearer zone surrounding them. It is an osteoid tissue that has already commenced to calcify.

The numerous spaces, containing medullary substance, in the spongy bone above the cochlea and the vestibule, are, like those lying further inward, completely free from any pathologic change.

On the posterior wall of the tympanic cavity, but even

more on the external and superior wall of the attic, great changes are visible in the bone. Especially in the anterior part of the attic, the cells adjoining the middle ear are entirely filled with dense fibrous connective tissue, and are separated from one another only by small plates of bone. These also consist for the most part only of osteoid tissue, that is lined at the periphery with layers of osteoblasts. In the superior layers, lying next to the dura, the bone seems only a little more altered, and only in a few cells are there dilated vessels, osteoid tissue and a rich cellular content. Scanty changes in the bone are found in the Glaserian fissure, in the bone of the tuba Eustachii, or in the mastoid process, although the bone in the region of the semicircular canals shows a sclerotic structure, and the lumen of the canals is very irregular, on account of excavations and small bony prominencies on the inner surface, i. e., shows changes of older nature. In the posterior part of the mastoid, in the bony walls of the cellular space, there is found a severe hyperemia with dilatation of the vessel channels, and osteoid areas with osteoblasts on the bony wall.

LEFT EAR.—In the internal meatus, the degenerated nerve bundles are less numerous and smaller than the right, but the hemorrhages are as profuse. The parts of the cochlea are well retained and show no variation; the vestibule and canals are similar to the right side.

MIDDLE EAR.—The lining of the cartilaginous tube is similar to the right; that of the bony tube is richer in cells and the vessels are over-filled with blood. Broad strands of connective tissue pass from the lining into the adjoining bony spaces, which also are partially filled with connective tissue and surrounded by pathologically altered bone. The internal layer of the drum is thickened and consists of sclerotic connective tissue, the mucous membrane covering both large ossicles is in places moderately inflammatorily infiltrated and adjoining these diseased portions of the mucous membrane are large spaces in the hammer which are filled with proliferated connective tissue and dilated vessels, and are bordered by new-formed osteoid areas and osteoblasts. The short process in places shows calcification of its cartilaginous layer. The anvil is not pathologic except for a

hyperemia of its bone vessels. In the middle ear, as in the right is found in places fibrinous inflammation of the mucous membrane, and this is especially plain in the region of the niches and on the floor of the tympanic cavity. The swelling and infiltration of the mucous membrane have not attained a very high degree, yet beneath the fibrinous exudate, especially in the niches, are band-like adhesions, which contain numerous spindle cells, as do the deeper layers of the inflamed mucous membrane. The same fibrinous inflammation is found in the spaces under the vestibule and cochlea which border on the middle ear, and which are lined with mucous membrane, while some of the smaller of these cells are filled out with a connective tissue rich in spindle cells. Cells filled only with mucus are also found. The sinus tympani is entirely filled with connective tissue, and its walls show a fresh hyperostosis of bone, which is found anteriorly only partially, but posteriorly in the whole periphery of the sinus; fresh areas of osteoid substance and layers of osteoblasts are also present.

The mucous membrane in the aditus and antrum is somewhat thickened, and some cells of the mastoid are partially filled with connective tissue.

The bone of the left ear shows, in general, the same pathologic changes as the right, yet there is a difference, since the left shows no changes on the external wall of the round window, but instead a larger otitic lesion in the anterior border of the oval window's niche. This extends between the periosteum of the middle ear and the endosteum of the sacculus rotundus, i. e., affecting the entire bone from within outward, anteriorly where it soon stops, at the plane of the passage of the facial canal through the bone. Posteriorly, it projects in the bone of the upper periphery of the niche of the round window only to the broadening out of the nerve of the utriculus, becomes smaller and smaller, extends more to the surface of the posterior superior periphery of the round window's niche, where it forms a small exostosis, and ends before the posterior margin of the oval window's niche. Histologically, this focus consists almost throughout of osteoid tissue, in which, centrally, in places, are remnants of old bone, and this is traversed by wider and narrower canals.

The former contain wide vessels, a delicate fibrillary network and large cells, resembling developmental cells; in places also long spindle shaped cells, and some round cells. At the border these canals contain loose osteoid areas of bone, and layers of osteoblasts, but at places are large multinuclear osteoclasts, especially at the borders of the healthy bone.

If we review the pathologic changes of the two temporal bones, we find an acute otitis media fibrinosa of mild degree accompanied by signs of a preceeding inflammation, such as the thickening of the mucous membrane by fibrous connective tissue in the middle ear as well as the adjacent cellular spaces, several of which are entirely filled out with fibrous connective tissue. The bone was changed in different ways.

At the necropsy, an extensive hyperostosis of the cranial bone, which reached a thickness of 12 mm. in the frontal bone, was found, and the bone of the pars petrosa, especially in the neighborhood of the semicircular canals showed a more sclerotic character. There were also acute inflammatory changes, partly in the ostitic foci in the niches, partly as a more superficial lesion in the external part of the attic. Furthermore, there was a periosteal hyperostosis on the internal wall and a sclerosis of the bone beneath the round window in the left ear.

While the first-mentioned changes of the bone and the hyperostosis of the cranium are of older character, the latter, which were especially prominent in the middle ear and the bone, are only weeks or months old, and are referable to an acute inflammatory process that probably appeared during pregnancy. I must leave it undetermined, however, whether all these changes refer their origin to the same cause.

REVIEW.

In the investigations reported, I examined 12 temporal bones of 7 patients, in all of which the same bone disease was found. In 5 patients, the process was found the same on both sides, in 2 I obtained only 1 temporal bone for examination, and therefore can give no report about the other

ear. 3 of the patients were males, 4 females. Their ages, at the time of death were 27, 28, 34, 58, 61, 63 and 76 years. The time of the disease, especially in the older patients, was much earlier and as much as this could be determined from the history, which just on this point was not always trustworthy, it seems that the disease appeared in all between 20 and 40, at the most 50, years of age. These results agree completely with those of E. Hardtmann,* according to which, in 22 cases collected from the literature, the disease began in the period between 20 and 50. The most began before this, in 8 between 30 and 40, and in 5 between 20 and 30, and if I add to them the 7 cases examined by me, I can say that of 29 cases, the disease began in 9 between 20 and 30, while in my three other cases, the process began before or after the 40th year. As the exact determination of the beginning is impossible at this age, and we can experimentally assume correctly a longer rather than a shorter duration, we will not err in the assumption that, in most cases, the time of the disease falls in the 20-40 years. Of the later cases, one of Siebenmann,† was 64 years old, and had taken sick many years before, and of two new cases of Scheibe,‡ one was 27 years old, and the other was a deaf mute 8 1-2 years of age. Therefore only the last case, of which I will speak later, shows a deviation from the above rule.

Characteristic of the disease is the appearance of peculiar, sharply circumscribed foci which are present in the bone either singly, or, usually, in the number of 2 or 3, seldom more, in greater or less extent.

Their origin can be traced especially in very acute cases, in which they are present in fewer numbers; thus I found in cases IV and V, as well as in right and left ear of case VII, and in left ear of case I, only 1 focus in each bone, which had its location 4 times in the anterior periphery of the niche of the oval window, and 1 in the external periphery of the niche of the round window. Two such foci in each bone were found in the right ear of case I, 3 in both ears of cases II and III, although here, there was, in addi-

*Zeitschrift f. Ohrenheilk. Bd. XXXIII, S. 103.

†Zeitschrift f. Ohrenheilk. Bd. XXXVI, S. 291.

‡Transaction of German Otological Society, 1901, S. 175.

on]to the foci in the niches, a third at the anterior periphery of the internal meatus and the cochlea; while in case VI, in which the disease in the ear reached the greatest extent, as many as 5 and 6 otitic lesions were demonstrable. In this case, the foci fused often, but by exact examination, especially by comparing the course of the bone disease with the other cases, it was still possible to demonstrate the same focal extension. A case of similar degree was reported by Siebenmann*, in which he was able to demonstrate 6 foci in each bone, which were almost all isolated from one another.

The disease in the bone, as in all cases in the literature, appeared always at the same places, and from these spread in certain directions in the bone. As I showed at the otologic meeting in Breslau,† it seemed to me that the bone disease began where larger or smaller vessels enter the bone from the periosteum, and that it extends later along these vessels. I came to this belief not only on account of examination of cases, in which the process had a very circumscribed position, but I convinced myself by injecting specimens of the temporal bone.

On the surface of the bone, the process usually attained only smaller extent, in that the periosteum showed localized thickening, and the vessel channels leading into the bone were widened. Only exceptionally, always in the foci in the internal meatus and in case II and III, also in the oval window's niche, was the surface of the bone ulcerated, uneven, with jagged processes, and in the internal meatus covered with scant fibrous tissue, while the ulcerated places in the middle ear were filled out with a thick fibrous connective tissue. In contrast to the mild changes on the surface, stand, usually, the great amplitude of the lesion in the depths of the bone, which in several cases affected the whole bone between the endosteum of the vestibule and cochlea on the one hand, and the periosteum of the middle ear on the other, where the process was not very extensive. I sometimes observed a tongue-like advance in the external cochlea wall, forward. I found the oldest changes in the bone

*Zeitschrift f. Ohrenheilk. Bd. XXXIV. S. 356.

†Transaction of the German Otological Society, 1901, S. 182.

usually at the point of origin of the focus, that is at the anterior border of the oval and the external border of the round window's niche, while the latest changes were usually at the internal periphery of the focus, i. e., near the endosteum of the labyrinth or at its anterior or posterior periphery. Thus it could be especially shown, in the focus in the niche of the oval window, that this usually extended forward, and only later posteriorly to attack the oval window's niche. It usually reached further posteriorly with its upper than with its lower periphery. The annular ligament, the base of the stapes and the membrane of the round window became involved secondarily in the diseases by its passing over from the labyrinths capsule.

An exception was found in case II, in so far that there were fresh changes in the bone immediately under the periosteum of both the round and oval window niches, and this can be explained only by the fact that there was a new disease of the same part of the bone, a relapse if we will not concede that the disease in this case began further forward on the promontory, which can be proven only by further examinations.

The arrangement as well as the extent of the individual ostitic lesions, showed a certain agreement on both sides, which can be explained only by assuming that the disease penetrated into the bone and increased by way of the vessels. The disease usually affected only the capsule of the labyrinth; the real membranous labyrinth was almost never involved: only the endosteum was somewhat thickened or calcified if the process reached so far. Only once, in a very severe case, was the external superior periphery of the ligamentum spinale greatly thickened; and, a few times, the bony surrounding of the spinal ganglia and the lamina spiralis of the basal turn were extremely calcified, and only in case VI were the dividing walls of the cochlear turns involved in the otitis. I often observed that the diseased bone projected in the form of an exostosis in the labyrinthal space; in case II were exostoses bilaterally on the anterior superior periphery of the stapes; in case III there was an exostosis at the oval window and a large exostosis at the left round window, projecting into the scala tympani. In case

VI, was a stenosis of the cochlear cavity in the apex and base, as well as the lumen of some of the semicircular canals by hyperostotic diseased bone. As the bone of the internal wall in this case showed an increase in size *in toto*, the lumen of the middle ear was thereby decreased. As an exception, in case II, there was in the end portion of the scala tympani new formed endosteal bone, which was attenuated by the ostitic process in the cochlear capsule. If this finding in the bone arose on the base of the same process as the otitis in the cochlear capsule, it must have happened in the beginning of the disease, which perhaps at that time was more severe.

The histologic changes shown by the diseased bone were very different at different places, and often all stages of the otitis were to be found near one another. In general we must distinguish the acute and the old changes. The former are found only in cases IV and VII, but the old changes were present in all cases. The fresh changes, as Katz* has already stated, resemble greatly the picture of otitis vasculosa of Volkmann. The hard bone of the pyramid was traversed by new-formed vessel channels which are formed by the old bones being melted away by the new vessels growing into them. How this happens, unfortunately cannot be determined with complete certainty in the comparatively thick sections of bone. In the small spaces, caused by disease process there was usually found only a dilated capillary with large endothelium, around which were some fibrillae and large developmental cells, sporadically some leucocytes and often lymphocytes. At the border of these spaces, the bone had a different aspect; very seldom were large multinuclear osteoclasts and lacuna formation found, as Siebenmann† has also stated. There were present, isolated, in some cases in larger and in others in smaller numbers, so for the dissolving of the healthy bone, other factors must be taken into consideration. The dissolving of bone by ingrowing capillaries was lately brought forward by J. Schaffer,‡ and the possi-

*These Archives Bd. LIII, S. 68

†Zeitschrift. f. Ohrenheilk. Bd. XXXIV.

‡Kölliker; Tissue Lore Bd. III, S. 673.

bility shown of osteoclasts developing directly from the cells of the capillary walls; it is probable that a similar process was present in the bone resorption in the cases described. I often saw also, as noted above, some cells of the otherwise healthy bone, at the border of the lesions, that had increased in size and had formed a small court around themselves, and, between these cells, a small canal had appeared as the first sign of the disease,

This process of vascularization of the bone has been described by Rindfleisch as canaliculation, and, as I will mention here, has been observed by Soloweitschik* in syphilitic ostitis of the cranial bone. It seems that the bone corpuscles do not play the passive role so often ascribed to them, but, as my specimens show, take an active part in the process. A new work by Kurpjuweit† also makes this seem probable.

With the formation of the vessels traversing the bone, the resorption process is usually ended, and then comes the new formation of bone and the retrogression of the vessel channels. The size of these channels, at times great cavities, depends on intensity of the cause at the bottom of the disease whereby the breaking down of the bone is sometimes very limited and sometimes extensive. The new formation of the bone comes about through osteoblasts which often lie in rows along the individual borders of the bone, but often are irregular, so that, as a result, the position of the bone cells in the new formed bones is a very irregular one. I advanced, above, the assumption that in exceptional cases the old bone cells play a role, since their increase in size and the partial absorption of the bone in their neighborhood is immediately followed by additions and deposit of calcium salts. By the new formation of bone, either the existing canals and spaces are caused to disappear, and thus a completely sclerotic bone is formed, or, as is usually the case, the spaces in the bone continue to exist, and their contents are changed into a scant medullary substance; or they disappear with the exception of a small amount of connective tissue at the borders, so that the spaces are almost empty. The bone in its neighborhood, which at first had an even more osteoid character,

*Virchow's Archiv., Bd. XLVIII.

†Virchow's Archiv., Bd. 163, S. 287.

stains deeper with eosin, becomes more and more calcareous, and is distinguished more plainly from normal bone. Its cells are usually larger, irregularly arranged, and usually no clear lamellar structure is demonstrable.

CAUSE.—The most important point in this disease of the bone is the cause to which its origin is referable, and in regard to that, the opinions of the authors are still diverse, and theories are advanced based partially on clinical and partially on pathologico-anatomic findings. Katz,* when he reported his first observation, thought of a rheumatic disease of the stapes—vestibule articulation, while, I† at my first observation, because I found a cicatrix of the drum also, assumed a sequel of an otitis media, which when it appears in the course of an acute severe infectious disease, e. g., typhus, runs a very severe course and easily passes over into the bone. After the examination of 4 other temporal bones of two cases, I‡ described, in the pathologic anatomy in Schwartz's Handbook, this disease of the bone as the cause of the bony ankylosis of the stapes, and claimed that the bony ankyloses of the stapes described by Toynbee, Voltolini, Schwartz, Moos, and Tröltzsch were caused by the same bone disease. There I said, also, that I had observed the same changes in the bone after purulent otitis media.

Politzer,§ who examined his specimen of ankylosis of the stapes histologically with this in view, and found the same disease of the bone, in spite of the large number of case examined by him, could not decide on a specific cause, and spoke of hereditary predisposition, puerperium, syphilis and gout, and in the latest edition of his text book, of ozena, also, as causes. He also accepted the view of Walb that the development of this disease is favored by anemia, mental affections and nervousness. In children up to 15 years, it is to be referred to heredity, rickets and scrofulosis.

*Deutsche, Med., Wochenschr., 1890, No. 40.

†Zetschrift, f. Heilk., 1891, Bd. XII S. 381.

‡Schwartz's Handbook, 1892, Bd. I. S. 248, & 258.

§Politzer, Zeitschr. f. Ohrenheilk. Bd XXV. S. 309, Lehrbuch, IV. Aulf, S. 263, and Beleuchtungsbilder der Trommelfells, 1896, S. 79.

Gradenigo,* considered this disease as a late manifestation of hereditary syphilis, and classes them, in the sense of Fournier, with the parasyphilitic affections. Siebenmann,† takes a peculiar standpoint: According to him, the process, which he calls a spongifying of the labyrinth capsule, arises from the zone between the primary endochondral labyrinth capsule and the secondary bone formed from the connective tissue of the periosteum.

Therefore, he will not designate this spongification as a simple otitis, but considers it rather as the last phase in a development which is not normal in the pars petrosa, but which is the rule in other bones, though in another form and at another time.

The foundations for this supposition of Siebenmann must be read in his article, and I do not consider them correct, but think that Siebenmann came to this conclusion only because in his first cases the process was very wide spread. Bezold,‡ and Scheibe,§ who in 1893 and later reported such cases, say, only, that the disease arises from the periosteum of the middle ear. Schwabach,|| in his work on the "Diseases of the ear in leukemia," described a case (XIII) in which, in addition to the changes caused by the leukemia, he found an otitic lesion in the cochlear capsule, concerning which he could not decide whether it was referable or not to the syphilis with which the patient was affected. Finally Katz,¶ has lately published a case, and in it has gone the deepest into the etiology of this disease. He assigns to the middle ear process, so often present, only a contributory value, and regards it only as an accidental antecedent, just as I/ did in my first case, and as I must even now agree with Katz. The chief factor in the origin of the bony disease, he

*Archiv., ital. di otologia. Bd. II S. 478.

†Zeitschr. f. Ohrenheilk., Bd. XXXIV. S. 356, XIII. internat. Congress in Paris, 1900.

‡Zeitschr. f. Ohrenheilk. Bd. XXIV. and XXVI.

§Transaction of the Otological Society, 1901, page 175.

||Zeitschr. f. Ohrenheilk. Bd. XXXI. S. 122. u. 145.

¶These archives. Bd. LII. S. 75.

/Zeitschr. f. Heilkunde. Bd XII. 1891, S. 381.

ascribes to constitutional or dyscrasic diseases of the body, among which he includes especially, (1) the rheumatico-gouty, (2) the scrofulous, (3) the syphilitic, (4) unnamed senile changes, (5) the neuroparalytic and trophoneuritic conditions. The latter and the rheumatico-gouty condition he considers the most important.

Through the examination of other cases of sclerosis, especially through the comparison of the finding in sclerosis with those in acute and chronic otitis media purulenta, I have more and more come to the conclusion that the bone diseases described is an entirely specific form of disease, whose cause I regard as syphilis. As the reasons for this supposition, I will advance the following: (1) In two of the cases examined histologically, viz., the case of Schwabach* and my No. IV, † syphilis was clearly present, as it was in two similar cases, examined only macroscopically, viz., one of Voltolini‡ and one of Downie,§ while in a case of Schwartzell, syphilis was very probably the cause. Therefore, lues was certainly the cause in 4 of the 30 cases, and almost certainly in 1. Even in the other cases, lues, although not proven, cannot be excluded with certainty. Thus in my case II, the affection began while the patient was in the army at which time syphilis is not rare; in my case VII the patient was a servant in a small inn in a street with a bad name; in case III, the patient was a single servant girl, with a daughter; and only case IV was a woman who had several children. Unfortunately in this case, the husband evaded every question about the ear affection of his wife, therefore in this case and cases I and V, I could obtain no information.

(2) A second reason, that speaks for syphilis, is the frequent beginning of the disease between the 20 to 40 to 50 year period: i. e., in a period in which syphilis is most frequently acquired, while other diseases are rare just in this

*Zeitschr f. Ohrenheilk. Bd. XXXI, S. 122.

†Transaction of the Otological Society, 1898.

‡Virchow's Archives Bd. XXVII, S. 163.

§Zeitschr f. Ohrenheilk, Bd. XXX.

||These Archives Bd. IV, S. 254.

period. The cases of Bezold, where the diseases began at 17 years, and of Scheibe, at eight and one-half, do not speak against syphilis, which in Bezold's case could have been acquired early, and in Scheibe's could have been inherited.

3. The histologic findings in the diseased bone also speak for syphilis. I found in my specimens a chronic ostitis which usually advanced from the periosteum to the bone via the vessels, and spread under the surface; which showed a complete chronic course, and can last 30 years and more; in which there are probably relapses of acute inflammation; in which suppuration and necrosis are absent or only exceptionally present; where no bacteria have so far been demonstrated in the diseased tissue as a cause; i. e., everywhere changes such as are observed in syphilis of the bone. The appearance of several foci in the bone is observed in syphilis. Chiari* found numerous foci in the long bones, and it is not unusual to find the simultaneous appearance of several lesions in the cranial bones. If all authors have heretofore ascribed to the temporal bone a certain immunity against syphilis (M. B. Schmidt †), this rests simply on the fact that these lesions have rarely been accessible to clinic examination and practically never to pathologico-anatomic. The rarity of syphilis affecting it is explainable by the fact that that external irritation is almost lacking, which often causes syphilitic diseases. Often an inflammation of the middle ear can give the impetus required for syphilis to awake here, and I can in this sense agree with the above opinion of Katz‡ that the middle ear process so often present has only the value of an exciting factor. The finer histologic processes, described above, are similarly often observed in syphilis. Thus Soloweitschik, states that the osteoclasts are seldom present in syphilis of the cranial bones, and that the canalization of Rindfleisch and the vascularization of Volkmann are the chief causes of the bone absorption. He also observed the increase in size of the bone corpuscles, and that they take an active part in the disease. Although these

*Vierteljahrssch. f. Dermatol. u. Syphilis.

†Ergebnisse der Allgem. Pathol. u. pathol. Anatomie VII S. 248.

‡These Archives Bd. LIII, S. 75.

changes are not characteristic of syphilis, still the agreement of my findings with those of true syphilis of the cranial bone is worthy of notice. If now we consider the end stages of the process, especially in a patient in which the disease has become wide-spread, e. g., in my 6th case, the increase in thickness of the bone, the hyperostoses with the almost complete porosity of the diseased place, and spongy exostoses in case I and others, show clear analogies with bone changes seen in syphilis. We are reminded of the increase in thickness present in the long bones, and the circumscribed projections with porosity and greater fragility of the whole bony substance, so that the only difference is that these changes are found in an unusual place, namely, the petrous pyramid.

It is still necessary to mention two circumstances that speak against these bone changes being syphilitic, viz.: the lack of distinct gummatous tissue, and the rarity of any destructive process, especially caries and necrosis of the bone. That these do not speak against syphilis, I can refer to Virchow*, who states that with an attenuation of the syphilitic virus, there usually arose products which often have only the character of a hyperplasia, and mentions as an example of this kind of syphilitic changes, the hyperostoses of the bones of the extremities, which, as I mentioned above in case VI, similarly develop in the temporal bone. Volkmann† also says that in addition to the more specific process of gummi formation, the syphilitic bone diseases show also only the picture of simple inflammation, which is apparently indistinguishable from the non-syphilitic but has individual characteristics at the most in the locality and the total habitus of the changes brought about. Soloweitschik‡ stated in his work on syphilitic cranial affections that caries and necrosis are not to be regarded as conditions belonging to the syphilis, but only as the possible sequel of the perostitis and otitis gummosa under especially unfavorable and accidental conditions. That other destructive processes were not entirely absent in my cases is proven by the specimens of cases

*Die Krankhaften Geschwülste S. 403.

†Handbook of Pitha, and Billroth. II. 2. Abt. S. 268.

‡Virchow's Archiv. Bd. XLVIII. S. 195.

I, III, II, and VI, in which are found defects in the internal meatus, and those of cases I and II, in which were found similar defects of the bone in the niche of the oval window. The former especially gives the impression of being healed gummatous periostitis and otitis. The second circumstance which speaks against this form of disease being a syphilitic one is the absence of other syphilitic lesions elsewhere in the body. If, now, we examine carefully the cases of sclerosis, reported in the literature, we do not find a complete necropsy given in the greater number of them, and even when it is given, and contains no mention of syphilis, this is really no proof that no syphilis had existed. It is precisely the osseous system alone that is most frequently affected by syphilis, while the internal organs and the skin show no lesions, and an exact examination of the bones at the necropsy is often not made. The diagnosis of previous syphilis is much more seldom made in the cadaver than it is observed in life. I would here like to mention that in some of my cases, in the post-mortem report is found the statement: Cranium thick and compact; and especially in case VII, the extreme hyperostosis of the cranium which caused a thickness of 12 mm. in the frontal bone, so that it can be supposed that this patient suffered from hereditary lues. Unfortunately, I paid too little attention at first to the presence in my specimens of a change that frequently is found in syphilis, viz.: disease of the vessels, and only later examined the sections with this in mind, and found that I could demonstrate in most of the cases endarteritic changes. I failed only in cases II and VII. In some, the changes might have been due to the age of the patient, and I will examine all new cases for these changes especially.

Of the other causes which could be assigned to the origin of this disease, acute and chronic inflammation of the middle ear must be mentioned first. Herefore I was under the impression that these under certain circumstances attack the bone in a milder form and cause these bony changes.

That, truly, disease of the bone accompanies acute and chronic middle ear inflammations, is proven by a number of pathologico-histologic investigations, in addition to numerous clinical observations. I* myself have proven that in

*These Archives. Bd. XLII. S. 128.

otitis media purulenta acuta the inflammation usually attacks the cellular spaces, pneumatic and medullary, that border on the middle ear and are in communication with them, and that accordingly as these spaces are developed differently in different people—above or below the labyrinth capsule—circumscribed inflammations and small empyemas appear in them, while the real labyrinth capsule shows only scanty changes in the larger vessel channels (sequelae of congestive hyperemias) and can be affected by the inflammation only secondarily from the middle ear itself, or the cellular spaces. At one place or another there is an erosion of the bone and a perforation into the labyrinth, and numerous examples of this kind have been lately described by Panse* and Manasse.† I myself have examined several such cases, although I have not described them fully.

Furthermore, I have described two forms of lesions of the bone that similarly are accompanied by hyperostosis of the internal wall and appear as a result of middle ear inflammation. In one‡ the hyperostosis of the promontory and the occlusion of the window arises through an ossifying periostitis; in the other§ through a superficial otitis, a form of disease which, in spite of its similarity to that accompanying the so-called sclerosis, differs in its extent, duration and sequelae.

A second form of inflammation of the bone, that could be used to explain the otosclerosis, is infectious osteomyelitis. This disease likewise enters into the bone via the vessels, and under certain circumstances can cause a return of the inflammation after the lapse of a large number of years, even 29. There also exist milder forms of this disease which Kocher and Tavel|| have described as proliferating forms (ostitis vasculosa, sclerotica, granulosa, and serosa). Such more chronic forms of infectious osteomyelitis have been

*These Archives. Bd. LVIII S. 184.

†Zeitschr. f. Ohrenheilk. Bd. XLIV. S. 41.

‡These Archives. Bd. LIII. S. 52.

§Ebenda. Bd. L. S. 242.

||Vorlesungen über chirurg. Infectiouskrankheiten, I. S. 145.

described by Garré,* Kocher,† Ehrlich‡ and Jordan,§ and Albert and Kolisko,|| in a work on osteomyelitis of the cranium directly make the claim that this in the temporal bone often causes an otitis media, and runs its course under the picture of this disease. Zeroni,¶ also relates a case in which, in addition to other osteomyelitic foci in the body elsewhere, there was also one in the bone of the external meatus, without involvement of the middle ear.

A circumstance which speaks very forcibly against the identity of our otitis with a milder form of this disease, is that, as most authors state, infectious osteomyelitis is a disease of youth alone, and that at the 20th year the tendency to this disease is almost entirely gone, while in otosclerosis the first appearance of the disease is observed almost exclusively in late life, from 20 to 50 years of age. Furthermore, even in the chronic forms of infectious osteomyelitis, formation of sequestra, although not the rule, is very frequent, as the observations of the above authors show. Finally in this disease, the cause of the infection, usually the staphylococcus is found in the bone, and can remain there many years; in the cases of otosclerosis heretofore examined no bacteria could be found as the cause of the disease.

Of other causes that must be taken into consideration in determining the cause of the described bone disease, I must mention the rheumatico-gouty diathesis, on which Katz lays great emphasis. In gout, we have a deposit of uric acid salts in the joints, the matrix of the cartilages, the capsules of the joints, the tendon sheathes and ligaments, while the bone does not take part in the disease, i. e., a process entirely different from ours which attacks the bone especially. Rheumatic disease of the joints conducts itself similarly, and,

*Beiträge zur klin. Chirurgie, 1893.

†l. c. S. 141.

‡Ueber latente Eiterherde im Knochen. Münch. Med., Wochensh., 1896.

§Ueber akute Osteomyel. Beitrag zur. klin. Chirurgie. Bd. X. S. 749.

||Untersuch über Osteomyelitis, Wien. S. 27.

¶These Archives. Bd. LIII. S. 316.

at the most, we could think only of arthritis deformans. Such a disease is denied not only by the histologic findings, as I convinced myself by an example that I was able to examine, but also by the non-involvement of the other joints of the body, a large number of which are usually involved in arthritis deformans.

That the puerperium has a causal relationship to otosclerosis is especially insisted on by Politzer,* and two of my cases, Nos. IV. and VI., seem to confirm this. It is well known that changes in the bony system not infrequently follow the puerperium, and Hanau,† has lately studied these changes. Siebenmann,‡ reports that Hanau examined his specimens of otosclerosis and gave his opinion that he could find no relationship or similarity to the puerperal bony processes. As well as I was able to understand Hanau's report on puerperal bone disease, and by my own examination of two cases of this disease, I am convinced that they are two entirely different conditions. A certain correctness cannot be denied to Politzer's opinion, as I can prove by several of my own clinical observations. Frequently pregnancy is accompanied by a syphilitic infection, and we know that in just such women syphilis is often more latent, not showing clear external symptoms. Likewise a hereditary luetic can acquire an otosclerosis as a result of pregnancy, an example of which is given by case VII., if the disease in the bone had not already commenced before pregnancy.

If we compare the results of the above investigations with our clinical experiences, these also strengthen the probability of such a cause for otosclerosis. Though it must be admitted that forms of tubal and middle ear catarrh, as well as mild forms of acute middle ear inflammation can cause a similar disturbance in hearing with the characteristics of a severe interference with sound conduction, still there are plenty of cases in which syphilis can be proven to be the cause. Of 116 cases of luetic disease of the ears that I col-

*Beleuchtungsbilder der Trommelfells, 1896, S. 79.

†International Congress at Rome. Vol. II. Page 148.

‡Zeitschr. f. Ohrenheilk. Bd. XXXIV.

lected in my treatment of the luetic diseases of the ear,* I found 24, that on testing the hearing, gave the picture of an extreme interference with sound conduction, and in these I had to refer the disturbance in hearing to luetic changes on the internal wall of the middle ear and in the window niches. In the histories of patients of our clinic in the last 5 years, I lately found 20 (9 men and 11 women,) in which lues could be shown to be the cause of the sclerosis, and 25 others (7 men and 18 women) in whom nothing was said about lues, but where no other etiologic factor could be found, unless a previous purulent catarrh of the naso-pharynx. Certainly, in some cases, lues could not be excluded.

Of the first 20 cases, 7 were from 21 to 30 years old; 7, 31 to 40; 3, 41 to 50; 1, 51 to 60. A boy of 7 years showed a considerable sclerosis on the basis of hereditary lues, and one 19 years old had acquired it 2 years before. Of the other 25, 4 were between 21 and 30; 11 between 31 and 40; 9 between 40 and 50; and one 52 years old.

SYMPTOMS.—As already stated, different diseases are classed as otosclerosis, according to whether it is defined from a clinical or anatomic standpoint. Here we can treat only of the symptoms of that case which shows by pathologico-anatomic examination the above described lesions.

DIFFICULTY IN HEARING is one of the most frequent symptoms, and frequently attains a high degree in a short time, especially if the windows are greatly affected, but under certain circumstances, especially at first, can be minimal or even be absent. If, as in my case VII, only an otitic focus is present on the lateral part of the niche of the round window. The same can be true if the focus in the oval window extends more anteriorly and does not attack the stapes and its articulation, a thing which seldom happens.

Testing in these cases will show little that is characteristic, as also in those very severe cases where there is almost complete deafness, is, e. g., my case VI. In the cases of medium degree, which comprise the majority, will be found those signs which Bezold designates as characteristic, for bony ankylosis of the stapes, viz., raising of the lower

*Haug's Sammlung klinischer Vorträge. Bd. I.

limit of the musical scale, negative Rinné and prolongation of bone conduction. While the first two signs are usually present, the last was absent in case III, and if I examine for this point the 20 cases of clinically proven luetic sclerosis, I find it noted only 3 times. Lengthening of bone conduction, by the method used by me, the small c tuning fork recommended by Lucae, is very seldom present. Where the explanation for this lies, must be shown by further examination. The value of Gellé's experiment for the diagnosis of stapes ankylosis, on which Block and his pupils* lay stress, must be mentioned here.

A second symptom, that often is present, SUBJECTIVE NOISES, is explainable in many cases, as I have already stated† by the extension over of the disease to the round window and the neighboring nerves, the basal turn and the cochlear spiral. The changes in the internal meatus and lamina spiralis must also be taken into consideration. DIZZINESS also can be caused by the spreading of the disease from the the oval window onto the point of passage of the nerves of the semicircular canal, and this symptom as well as subjective noises can disappear after cessation of process. Diseases of the endosteum, as Siebermann state, can cause a decrease in the pressure of the labyrinthal fluid and thus symptoms of irritation, as probably happened in my case IV as a result of the closure of the aqueductus cochlearis. The appearance of the drum described by Schwartze is characteristic for ankylosis of the stapes, whose relationship to the bony disease of the promontory I showed in my pathologic anatomy, is present in those cases in which the drum has not been rendered less transparent through previous diseases. It can also be present where a hyperemia of the promontory is present as the result of a catarrhal process.

It is not difficult to make a diagnosis if the cause, syphilis, is known, but when this is not, it may be difficult to differentiate it from other processes that cause obstruction of the niches and stapes ankylosis. As such processes, that can be confused with the first form, and that lead to non-

*Stern, Immobility of the stapes, Wiesbaden 1903.

†Schwartze's Handbook, Bd. I, S. 248.

syphilitic sclerosis, can be mentioned catarrhal inflammation of the tube and middle ear, which extend from the nose into the ear, and secondly, certain forms of middle ear inflammations which run their course without severe subjective symptoms and without perforation of the drum, and can lead, in a short time to severe impairment of audition by organization of the exudate in the niches of the windows. The diagnosis of these forms from the luetic sclerosis is made by an exact history of the patient, and especially by the fact that in the catarrhal forms, exacerbations may often appear from external factors, catarrh of the nose, etc., and amelioration is accomplished by the catheter and treatment of the catarrh.

The diagnosis of luetic sclerosis as a syphilitic periosteogenetic otitis and osteomyelitis from the other forms of syphilitic disease of the labyrinth can, under certain circumstances, be very difficult if not impossible. This is especially true for the endosteogenetic syphilis of the labyrinth, the endosteal labyrinth syphilis that causes a filling of the inner space of the labyrinth with connective tissue (Manasse)* or even bone (Downie),† probably case III of mine. The diagnosis of nerve syphilis, which is more frequent, is much easier and Manasse and Politzer‡ have made histologic examinations thereof.

The treatment of luetic sclerosis can be only antiluetic, although I must admit that we often start too late. That we can expect from it something under favorable circumstances, is vouched for by the fact that the iodides have for a long time been in favor in the chronic forms of progressive deafness. Against the fixation of the sound conducting apparatus, we can use the mechanical treatment from the external auditory meatus, which has the purpose of restoring mobility. Some of my experiences have taught me that some good, moderate though it be, can be so accomplished.

*Zeitschr. f. Ohrenheilk. Bd. XXXIX S. I.

†Ebenda. Bd. XXX.

‡Lehrbuch der Ohrenheilkunde.

L.

OTITIC SEROUS MENINGITIS, LUMBAR PUNCTURE
—RECOVERY.

FRANCIS H. HUBER, M. D.

NEW YORK.

Ida B., two and a half years old, born in New York, of Russian parentage, was admitted to the children's ward, Beth Israel Hospital, January 8, 1903. No history of tuberculosis in the family. History of insanity in grandmother, uncle died insane, aunt suffers from epilepsy. Father alcoholic.

PERSONAL HISTORY:—Was born after difficult labor, though instruments were not employed, cyanosed for several days, cry feeble. Breast-fed for nine months, began to walk at 17 months. For more than two years has had a foul smelling discharge from the right ear, the original cause of which could not be made out at the time. No history of any eruptive fevers. A subsequent examination revealed large adenoids.

PRESENT HISTORY:—Fourteen days before admission, the child became restless, began to cry a great deal and acted as though greatly frightened. Four days later began to have convulsions. The seizures were general and occurred every thirty minutes. The attacks were characterized by a loud cry, the child would fall to the ground suddenly. The movements were both clonic and tonic and lasted about three minutes. Of late, the attacks have increased in frequency, but are of shorter duration. Occasionally vomiting occurred after an attack, at other times the child would cry and fall asleep.

The convulsive attacks occur at night as well as during the day and are brought on by any external source of irritation. On admission: General nutrition good. Face suffused, expression dull, child apathetic and in a semi-comatose state. Extremities cold and blue. Surface generally mottled. Tongue coated, moist. Eyes convergent strabismus, moderate

lateral nystagmus. Pupils dilated, right more so than left. Under light test contract slowly and to moderate extent. Then slowly dilate again, and again contract. Tache cerebrale easily produced. An offensive purulent discharge from the right ear, no tenderness over the mastoid region or side of skull. No edema of this area. Pulse rapid, no irregularity or intermission. Reflexes in general exaggerated. Moving the patient or attempts at feeding were followed by convulsive seizures. Lumbar puncture proposed for diagnostic purposes, could not be carried out as the violent character of the attacks brought on in attempting to put the child in position, interfered with the necessary manipulations.

The neuropathic family history of insanity, epilepsy, etc., in the parents and near relatives suggested a condition of status epilepticus. The latter, however, could be excluded from the history of the case, and the fact that the convulsions dated back only two weeks. Careful inquiry failed to elicit a history of a fall, blow or traumatism of any kind. By exclusion therefore we were compelled to look upon the chronic suppuration in the ear as a probable etiological factor, though no focal symptoms were present. The statistics of Pitt estimate that 5 per cent. of all cases of meningitis are of otitic origin. Dr. W. Freudenthal, to whom I herewith extend my thanks for his wise and conservative counsel, was requested to examine the patient.

REPORT OF DR. WOLF FREUDENTHAL.

"On January 10th, 1903, I saw the above mentioned child at the request of Dr. Francis Huber. She had had convulsions, which were increasing in number and severity, the pulse was getting irregular and the whole aspect of the case appeared worse than at the beginning. There being a suspicion of mastoiditis I was called in. After removing from the right ear the offensive discharge that had lasted for the past two years, an exceptionally large perforation of the drum membrane could be seen, out of which pus oozed. There was no swelling over the mastoid, no tenderness or pain over any part, the temperature was 100, pulse 130 and the child cried incessantly. Besides it had had seven attacks during the past night and eight during the day. As the mastoid

symptoms were not very well marked, it was thought there could be no risk in waiting 24 hours longer. At the end of this period the attacks had increased in frequency and the general prostration was more marked. The general belief of all concerned in the case now was, that the only salvation for the child could come from the opening of the mastoid. At 2:30 P. M., on January 11th, the operation was performed in the usual manner. After chiseling away all the diseased bone, the dura mater was seen to bulge outward and it was apparent that there was pressure from within. I hesitated however, to withdraw any fluid, because an infection was possible and surely not desirable. The wound was packed and at 4 P. M. the house physician, Dr. Nisonoff under the supervision of Dr. Huber withdrew about 30.0 grammes of spinal fluid by means of lumbar puncture. Within the next two hours the child had three convulsive attacks. Six hours later twitching of the extremities set in beginning at 12 midnight and ending at 1 A. M. She became quiet after repeated hypnotic medication. Another such severe attack commenced at 4:45 in the morning and lasted three-quarters of an hour. At noon the next day (12th) 10.0 grammes of spinal fluid were withdrawn, after which the symptoms slowly but gradually improved and the child was discharged on February 2, 1903, i.e., 22 days after the operation was performed."

When seen subsequently about two months later she was bright and active, running and playing, presenting no evidence of her severe illness.

In the treatment of the case there is no doubt in my mind that in spite of the non-existence of any local evidence of mastoid trouble, the operation was justified. In addition to getting rid of the local diseased focus we were enabled to eliminate a possible pachymeningitis and at the same time relieve the pressure to a limited extent. In resorting to lumbar puncture an accidental infection, immediate or secondary, of the cerebral meninges was avoided. The favorable therapeutic effects following the withdrawal of the cerebro spinal fluid were manifested in a very few days. On the 14th the child was still somewhat restless. On the 16th it was bright, took interest in its surroundings and could with difficulty be kept in bed.

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LI.

PRIMARY EPITHELIOMA OF THE NASAL FOSSAE,
WITH REPORT OF CASE.

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The malignant growths in general do not occur very often in the nasal fossae, but of all, carcinoma is the least frequent.

Until recently but a few cases have been recorded in the medical press. For 1892 Dr. Dreyfus,* after a thorough research and study of the subject, reported only 13 cases in his very instructive treatise, "The Malignant Epithelial Growths of the Nasal Fossae."

According to his investigations the first case was reported by Robin in 1852, "Hypertrophie Glandulaire." Some other authors believe there were cases reported at an earlier date; we find for instance in the statistics of Prof. Gibb,† a case reported by Earle as far back as 1827. Nevertheless I consider the claim of Dreyfus to be more correct and reliable, because he considers only cases the diagnosis of which have been confirmed by a microscopic examination. In the last ten years we find single cases of primary carcinoma of the nose more frequently reported, but the total number of all reported cases is small enough to demonstrate that primary carcinomatous affection of the nasal fossae is of very uncommon occurrence.

In the year 1900 Prof. Kuemmel‡ found in the literature not more than 40 cases. Two years later Prof. Gibb in the

*Dr. Dreyfus. Wiener Med. Presse, 1892.

†Dr. J. S. Gibb, "Malignant Dis. of the Nose," N. Y. State J. of Med., page 56, 1902.

‡Kuemmel: "Die bösartigen Geschwülste des. Nase."—Handbuch der Laryng. und Rhinolog.—Heymann, Vol. III, 1900.

above quoted treatise reported 78 cases, but if we consider those cases only which were confirmed by microscopic examination, this number would necessarily be considerably reduced. In my opinion, the total of all cases, including the ones reported last year,* is between 60 and 70.

The question as how the primary carcinoma of the nasal fossae originates is as yet but little understood. Whether the benign tumors may become malignant is an open question upon which most authors differ; some believe for instance that polypi can change under certain conditions into carcinoma. As a proof of the assertion they point out the fact that in some cases of carcinoma of the nasal fossae polypi have also been found.

Prof. Kuemmel, Findert and others believe that the coexistence of these two growths does not prove that one growth changes into the other; they may develop entirely independent of each other. If any such transformation takes place at all, it is more likely to occur in regard to papilloma and adenoma.

But of all cases of primary carcinoma of the nasal fossae which have been reported, there is to my knowledge only one case where a change from a benign into a malignant tumor has been more or less proven—this is a case of adenocarcinoma reported by Dr. Cordes,† which changed from a pure adenoma.

In regard to this kind of tumor, Prof. Kuemmel says: "The distinction between this kind of adenoma and carcinoma is hardly to be noticed; being of the same histological structure some of them may conserve their benign character, while the others may turn earlier or later into a distinctly malignant tumor."

*S. Citelli and N. Calamida, *Beitrage, zur Lehre von den Epithelioma der nasenschleimhaut*. Arch. f. Laryng., 1902. B. XIII., Heft. 2. Wm. Darnal. "Prim. carc. of the Nasal Chambers."—*Jour. Amer. Med. Assn.*, Chicago, 1903.

†G. Findert: "Einige Bemerkungen ueber Malig. Nasen geschwulste."—*Arch. f. Laryng.*, Vol. V., 1896.

‡Dr. Cordes: "Das Adenocarcinom., Berl. Kl. Wochenschrift, VIII, 1903.

As to the seat of carcinoma in the nasal fossae authors differ in their opinions. Some mention first of all the upper part of the septum, then the inferior turbinate and others the roof of the nasal fossae (Kuemmel, Finder), while others (Cordes, Citelli and Calamida and others) on the contrary, point out the mucous membrane of the ethmoidal cells as the seat of the growth.

The nature of the carcinoma does not depend on its seat. Primary carcinoma of the nasal fossae may be classified as follows:

1. Cylindrical Epithelium Carcinoma.
2. Flat-Epithelium Carcinoma (Epithelioma).
3. Adeno Carcinoma.

The frequency of these different forms occur in the same order.

It is unnecessary here to emphasize how important it is in case of carcinoma to make a positive diagnosis early, which can be attained only through a microscopic examination.

The symptoms in general do not give a complete idea of the affection. Obstruction of the nose, which depends entirely on the seat and size of the tumor (it is missing in my case), is not at all characteristic of carcinoma as such, because it is apt to occur in cases of benign tumors as well as in some other pathological conditions of the nasal fossae. More characteristic is the sero-sanguinolent secretion.

Some authors (Cordes and others) consider the hemorrhage a constant symptom of malignant tumors of the nasal fossae in general; in fact, it is very important for differential diagnosis between sarcoma and carcinoma, for in most cases it is a positive and characteristic symptom of sarcoma. In carcinoma the hemorrhage is insignificant and secondary; a result of the ulceration on the surface of the tumor.

A very important symptom, which in some cases (at least in mine) is the most prominent in the entire symptom-complex is pain, which like neuralgic pain, according to the seat of the growth, irradiates in different directions, and is at times very intense. Necrotic processes with ulceration and slight hemorrhage occur in most cases, but hardly, if at all, cause a deformity of the surrounding bones, which is the rule in cases of sarcoma.

The age of the patient is also very material for the differential diagnosis between sarcoma and carcinoma, the former finding its victims in youth, the latter occurring with very few exceptions in advanced age.

The duration of the disease is an average of 1-3 years, although there have been cases of 4 to 7 months duration reported,* and others of 11-12 years.†

One of the most characteristic features of carcinoma of the nasal fossae is the lack of metastatic processes. If at all, these cases show a very slight disposition for infiltration of the regional lymphatic glands, while in cases of sarcoma it is hardly ever missing.

The *prognosis* of carcinoma of the nasal fossae is in general a very bad one. Almost all the cases operated upon as well as those which were not have been followed by death. The only lucky exception is the case of Dr. Cordes (adenocarcinoma).

As to the treatment there is not much to be said. According to the above quoted reports the results of radical operation are far from encouraging, still the radical operation remains the only means of treatment. In cases where an early diagnosis is made and where therefore the malignant process has not developed too far, better results may probably be achieved. In such cases a radical operation is to be considered.

The following case has been for some time under my observation.

W. F., 58 years old, family history good, in appearance strong and robust, was operated upon years ago for some rectal trouble (periproctitis). About a year and a half ago he suffered from an attack of scarlet fever from which he recovered very soon, but which evidently left some nose trouble. A short while after he noticed in the left nasal fossa a "pimple," which caused him quite an acute pain. Since that time he consulted various physicians but the pain did not yield to treatment. In the beginning of October the patient came to me.

*Douglas, Wm. Darnal and Pepper and Shakespeare, Philadelphia Medical Times, 1879.

†Newdorfer and Hopkins.

Status praesens: nothing is to be seen on the nose externally, except the frequent dropping of a sanguinolent secretion from the left naris. Free nasal breathing on both sides—no signs of any obstruction of the nose. Nasal examination shows the presence of a slight septal deviation on the left side, otherwise normal condition as far as the turbinates on the sinuses are concerned. On the lower part of the septum on the left side not far above the junction of the skin and the mucous membrane, a tumor can be seen about one-half cm. high, 1 cm. wide and 2 cm. long, of dark red color with an ulcerated surface. The appearance of the tumor as well as the peculiar pain which the tumor caused led me to believe it to be a carcinoma.

A piece of the tumor was microscopically examined and the report received reads as follows: "Sections show epithelioma with numerous pearl nests and multiple mitoses." The sections leave no doubt as to the nature of the tumor: masses of squamous epithelium of different shapes, formation of cell-nests of irregular forms, in many places the so-called epithelial pearls, which under close observation show the well known onion-like form of concentrically arranged cells.

As the tumor was absolutely circumscribed and attached to the cartilaginous part of the septum, I thought the best course of treatment would be the following: After an application of cocain and adrenalin I removed the entire growth with the scissors and snare, and then curetted the entire base of the tumor and a part of the surrounding healthy tissue. To avoid subsequent hemorrhage I applied chromic acid upon the operated surface. The wound did not take long to heal and ten days later the part of the septum which had been operated upon was as smooth and normal as the rest of the septum, and there was no pain. I have seen the patient a few times since, but could not detect any thing abnormal, or any recurrence within the nasal cavity.

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LII.

MASTOID DISEASE AND CEREBELLAR ABSCESS.

BY SEYMOUR OPPENHEIMER, M. D.

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The importance of encephalic pus collections in their relation to the diseases of the temporal bone, in which purulent inflammatory changes play an essential part, can only be estimated by the amount of destruction wrought in the brain tissue and adjacent structures and by the lethal issue resulting in the absence of prompt recognition and surgical relief. While various portions of the brain may be the seat of abscess formation, the temporosphenoidal lobe and cerebellum concern the otologist more than any other portions, as these areas are the most frequent site of secondary infection from the temporal bone. Cerebellar abscesses as far as their primary origin is concerned, may be classed into two groups, those due to infection by way of the labyrinth, through the petrous portion of the temporal bone and a second class in which the infection is derived from the mastoid process. The latter is the form which I desired to consider here.

The location of the pus collection in the cerebellum when derived from the original mastoid focus, is practically more or less constant in the anterior portion of the lateral lobe and on the same side as the area of bone necrosis, although in rare instances, multiple, discrete abscesses have been found on autopsy. Moos records a singular case in which the cerebellar abscess was on the opposite side to the diseased ear and was also associated with a cerebral abscess on the same side as the ear affected. The source of infection, if far back in the mastoid process, will produce the brain

lesion in the posterior part of the cerebellum, the direction in which the ear disease may be propagated varying as shown by Pitt, with the position of the sulcus for the lateral sinus.

While the groove varies considerably in different cases, in some temporal bones it is found well forward and in very intimate relation with the mastoid cells, so that the presence of a necrotic area in this vicinity, would greatly aid in facilitating the spread of the infection backward toward the cerebellum. For should this slight barrier break through and an extension of the purulent process take place through the inner wall with a circumscribed meningeal infection, this portion of the encephalon will inevitably be the seat of the pus collection. The sigmoid groove thus plays a prominent part in the conveyance of the infection, although the osseous lesion as regards its primary inception is less directly in relation with the abscess cavity than is usually the case with cerebral abscesses.

The thickness of the entire mastoid wall also acts as a restraining factor, particularly in children, the rare occurrence of cerebellar abscess in the very young from this locality, depending as shown by Gruber, upon the fact that the bony material intervening between the mastoid antrum and the cranial cavity, exists in relatively larger amounts in early life, than at a later period. But at any age, should this intervening layer of bone undergo destructive changes as the result of a mastoiditis and the suppurating process convert the sinus into an abscess cavity, the chances are largely in favor of a subsequent cerebellar abscess provided the patient does not succumb to the sinus phlebitis or infective meningitis. The brain abscess however, does not in all cases necessarily follow the mastoiditis directly, but may arise from an infective sinus thrombosis or an epidural abscess.

The large proportion of the cases of this class not being dependent upon an acute ear affection, as cerebellar abscess from this cause is very rare, but rather from an exacerbation of a chronic or latent focus of infection in the mastoid cells, which has been incited to renewed activity by some recent irritation. Such a case being reported by Kirkland in a boy of sixteen years, where the aural trouble has existed for three years. There was intense pain in the left ear, the tempera-

ture was 103° F., the mastoid was swollen and when the latter was opened it was found to contain but a few drops of pus. Severe occipital pain, left optic neuritis, vomiting, giddiness and drowsiness with a normal temperature and pulse then ensued and the skull was trephined one inch above the suprameatal wall with a negative result. On the following day the skull was again trephined over the cerebellum and a half dram of foul pus was evacuated, with the ultimate disappearance of the bad symptoms.

In other cases the inner mastoid wall may remain perfectly intact and the infection will travel through the small channels in the bone by way of the minute veins running from the surface of the cerebellum to the lateral sinus and from this location to the mastoid interior. The following case in which the infection was traced directly from the mastoid cells, resulted in a successful termination after operation :

L. G., male, aged thirty years; has had otorrhea of the right side for ten years, but no serious symptoms were complained of until one week before he was first seen, when giddiness and pain over the mastoid region developed. The mastoid was swollen and tender, with bulging of the wall into the canal and usual signs of a chronic otitis were present with a perforation in the posterior inferior segment of the drum membrane. He also complained of a gradual loss of weight and there was facial palsy on the same side as the mastoid disease. Anorexia, constipation and foul breath were also present, with beginning optic neuritis. Vertigo was the most prominent symptom, while nausea and vomiting were also occasionally present. Intense pain was complained of over the occipital region on the right side, with sluggish reaction to light of the dilated right pupil, and the temperature was 97.2° F., while the pulse varied between 60 and 70. Physical weakness was extreme, the gait was staggering, there was weakness of the right arm, exaggerated knee-jerk and the patient lay on the left side.

Cerebellar abscess following mastoid disease was diagnosed and immediate operation was advised and consented to by the patient. Under ether anesthesia and with the usual aseptic precautions, mastoid operation was first performed and considerable greenish pus and necrosed tissue was found

occupying the mastoid cells, while a minute sinus, with a corresponding small area of necrosis, led into the sigmoid groove. This was removed and the sinus was found to be somewhat discolored but otherwise healthy, while the dura was bulging into the wound. The brain membranes were opened and a grooved canula was plunged into the cerebellum, when about half an ounce of fetid pus was evacuated. Introduction of the encephaloscope showed a firm, resisting, limiting membrane. The abscess cavity and mastoid wounds were gently washed out with a 1:5,000 bichlorid solution and a gauze drain inserted. The temperature ran an erratic course for several days, but at the expiration of a week all of the severe symptoms had disappeared. The abscess cavity continued to discharge for two weeks when it ceased and the patient rapidly made an uninterrupted recovery.

The frequency of cerebellar abscess, as compared with the intracranial complications of mastoid disease, is but small; Barker believing, from his experience, that less than a tenth part of the ordinary complications of aural disease consists of this form of pus collections. While Gradenigo in a study of 68 cases of mastoiditis, found endocranial complication in twelve and of these but two were cerebellar abscesses. As regards the proportion of cerebellar to cerebral abscesses one may safely say that of all encephalic pus collections of otitic origin at least 25 per cent. are located in the cerebellum and that the majority of these are in the anterior surface of this portion of the brain, in immediate proximity to the sigmoid groove. Körner, in one hundred cases of brain abscess, found the cerebellum alone involved in 32, while in six cases both cerebrum and cerebellum contained a purulent focus. He found twice as many in males as in females and also agreed with the majority of observers that they were more common on the right side. Allport, in 169 cases, found 31 in the cerebellum; Jansen, in 16 otitic brain abscesses found 9 in this locality, a quite unusual experience; while Picque and Ferrier, in 119 instances found 24 in the cerebellum, 4 in both this locality and the cerebrum and 1 in the peduncle. Barr, in 76 cases, found abscesses 13 times in the cerebellum. In children as before mentioned, it is quite rare in this location, although one may be safe in estimating the per-

centage here as compared with all encephalic abscesses, at from 15 to 20; the greater development of the pneumatic spaces in the adult, increasing the predisposition to cerebellar abscesses as a complication of mastoiditis.

The symptom-complex of cerebellar abscess, while irregular and at times far from characteristic of the lesion present, may in some cases allow of the grouping of the symptoms, so that four stages of the process may be fairly well distinguished. The first commencing with the symptoms signifying the extension of the infection from the mastoid process to the cerebellum and classified as the initial onset of the local infection. This is followed in the majority of cases by an arbitrary second stage characterized by mild discomfort in which the disease remains latent and usually is not recognized. The third stage supervenes at a longer or shorter period of time and the symptom group of this period is characteristic of a severe otitic intracranial infection, sometimes with symptoms pointing to a localized cerebellar abscess, while more frequently, the pus collection is only localized after the parts have been explored by operative procedure. When, however, this stage is not recognized and the intracranial condition is allowed to continue without surgical relief, the fourth or terminal stage of the disease is an essential sequence, manifested by exhaustion, coma and the gradual death of the patient, or by sudden death from rupture of the abscess into some vital portion of the brain axis.

The initial or formative stage of cerebellar abscess, presents no definite signs as regards localization of the disease, but the usual symptoms present at this time are those of an acute extension of the mastoid suppuration to the intracranial contents and are usually more characteristic of a sinus thrombosis or a localized area of meningitis, than of a cerebellar affection. A temperature that is already high as a result of the preexisting mastoiditis, becomes a degree or more so, the pain over the mastoid region becomes more severe and may extend back over the occipital region, while vomiting may occur once or twice and then disappear or the symptoms may increase in severity and operative interference be indicated on account of the associated morbid pathology. In a large majority of the reported cases, however, the onset of the

intracranial complication if acute, as here described, was unnoticed, or the infection was gradual, probably covering a period of many weeks or months and passing into the second or latent stage, with a subsidence of any of the acute symptoms that might have been present.

Even more so than in the first stage are the symptoms at this time indefinite and often not of sufficient prominence to attract attention to the aural disease as the cause of the gradual failing in health, with frequent periods of intense headache, varying anorexia, more or less depressed mental condition and fairly well marked aprosexia. If a thorough examination were made at this time, an intracranial lesion at least would be suspected, but unfortunately such is not the case and if the patient seeks advice at all, he is usually treated for some vague general condition and an opportunity is lost which cannot be regained. Such a case came under my direct observation, several years ago, in a young man of twenty years. He had been in previous good health except for a suppurating right otitis which discharged at irregular intervals, but not enough to cause him any concern. Then the mastoid became painful and swollen, but under local medical treatment by his family physician, soon disappeared and nothing further was thought of it. It was noticed, however, that he gradually began to lose weight, to complain of frequent occipital headache and to have attacks of nausea and vomiting. His mental condition became dull and he would deliberate in answering even a simple question for several minutes before he gave a reply. This continued for several months, during which time he was taking medicine for a supposed gastritis and finally the symptoms enumerated became so marked, that he was referred to a hospital for a further study of his condition. Seventy-two hours later he died from a large cerebellar abscess which had ruptured into the ventricle, as shown by autopsy.

The third stage like that of the others, of course presents no fixed boundary lines, the transition from one to the other being gradual and usually not recognizable, but for purposes of study, these cases admit of some differentiation of symptom groups with their pathologic stages. At this time the full complement of symptoms have become well developed, and,

according to v. Bergman, can again be profitably subdivided into three groups: the general symptoms; those due to pressure and local pathologic changes; and the localizing symptoms.

Of the general symptoms physical weakness usually becomes more pronounced as the abscess develops and the muscular asthenia is especially marked on the same side as the pus collection, being chiefly confined to the arm. The muscular weakness may also be evident by the conjugate deviation of the eyes toward the unaffected side from asthenia of the ocular muscles and not from direct special nerve involvement. This muscular atony, whether local or general, develops most insidiously and may be the first symptom noted; its importance in connection with other symptoms being considerable as indicative of a brain abscess. A gradual impairment of the appetite, sometimes amounting to complete anorexia, may also be present, but in conjunction with a coated tongue and a peculiar yellow color of the skin, is indicative only, like the majority of the symptoms, of a septic intracranial condition and like those present in many grave diseases, is dependent upon the gradual absorption of septic material and its resultant action on the general organism.

The symptoms due to pressure and local pathological changes vary in almost every individual case, but the presence of headache is fairly constant in all, usually being the earliest and most conspicuous symptom. It is occipital in location and while very intense, is increased by pressure of percussion especially over the region of the abscess. Generally constant, at times it is not so severe, but again becomes almost unbearable. Rarely it may involve the frontal region and then of course, greatly interferes with the recognition of the pus collection, but as a rule it is situated in the former position, being duller in character and more limited in extent than that due to other intracranial lesions. Sometimes it may be the only practical symptom of importance for a time, as in a case reported by Heiman, of a man of twenty-four years, with sudden onset of a headache increasing in intensity, following the suppression of an aural discharge. No characteristic symptoms were present but in addition to the headache he had some fever and pain

in the ear. Death occurred within twenty-four hours after the beginning of the headache and the autopsy showed a ruptured cerebellar abscess in the process vermicularis.

Nausea and vomiting are almost always present but are not necessarily characteristic of cerebellar abscess. In connection with other symptoms and especially with unsteadiness in gait, vomiting may assume more or less of a characteristic aspect and then depends to a great extent on pressure exerted upon the middle lobe of the cerebellum. In connection with dizziness, vomiting has also been of great value in making a diagnosis as shown by Green and Crombie. Vertigo is present in nearly all cases and may be associated with a loss of the sense of equilibrium, the patient presenting a staggering gait. Or in the sitting posture the head may fall forward, then turn toward the unaffected side and continue to move with pendulum-like oscillations, this condition is extremely rare however and in such cases, the vertigo is a prominent symptom, being constantly kept up by the head movement.

The mental functions are usually disturbed and at first somnolence is common, but may be in isolated instances replaced by a condition of sleeplessness, this being the only symptom for which the patient seeks advice. The mental state may often furnish valuable information especially when the patient is irritable at times, while at others he may be inattentive or the hebetude as the disease advances, gradually giving way to stupor. Sluggish cerebration may be quite noticeable and the peculiar condition may exist in which, if a simple question be asked the patient, he will not appear to have heard it and no notice will be taken for a time, then in a few minutes he will begin to answer slowly and deliberately, dwelling carefully on each word, but the final answer will be correct.

The temperature at this stage is of much value as a symptom of brain abscess, but gives no information as regards the site of the pus collection. Oscillations of the temperature are as a rule not present, and it remains fairly constant at or near 99° F, after the initial rise due to the primary extension of the disease to the cerebellum. Very often it is normal or subnormal and when the latter is continuous in connection

with an aural lesion, the presence of intracranial abscess may be fairly well assumed. In some cases the diagnosis in great part will hinge on this symptom, a case in point being that seen by Morris, where the low temperature in connection with a subnormal pulse rate, led to the diagnosis of brain abscess, but precise localization was impossible. The autopsy showing a cerebellar abscess on the same side as the diseased ear. The pulse is also slow, regular and of fair volume and remains unchanged in uncomplicated cases, but it may be normal and occasionally become intermittent. An instructive case in this connection being seen by Schwartz, in which the prominent symptoms were intense, intermittent pain in the occiput, nausea, vomiting, subnormal temperature, irregular pupils and a rapid pulse of 100 to 150. No operation was performed, as the rapid pulse was considered incompatible with a cerebellar abscess, but the necropsy showed an abscess in the right cerebellar hemisphere, with circumscribed leptomeningitis and pachymeningitis interna and the cause of the rapid pulse rate was readily explained by the finding of a chronic endocarditis. Respiration is usually regular, but often slower and more shallow than normal.

Of the minor symptoms of this class, which are variable and may or may not be present in the given case, cervical contracture is one of the most frequent, the muscles of this region may also present varying degrees of rigidity and from the same cause, clenching of the teeth may occur infrequently. The tongue may be protruded toward the unaffected side, making the speech slow and indistinct, if it be swollen. Rigors occur infrequently, the bowels are constipated and the knee-jerk may be increased on the same side as the brain lesion. Walker reporting a successful case following operation, in which the only characteristic symptom was the increased knee-jerk.

The localizing symptoms are of little value in this affection, although Burnett states that double optic neuritis is likely to be present, because the abscess readily interferes with the circulation of the cavernous sinuses and the ophthalmic veins. Very often this may be the only positive symptom, but again the eye-grounds may remain normal even in fatal cases, as has been shown by Woodward. Hansberg believes that lo-

calization of a cerebellar abscess is almost impossible from focal symptoms, but he considers bilateral choked discs as the only sure sign, while Gradenigo states that lesions of the optic papillae are observed in about one-half of these endocranial complications and they should be carefully sought for, as they are frequently the first and only indication of intracranial involvement. He further states that the papillitis affords no information in regard to the location of the lesion.

Nystagmus and paralysis of the sixth nerve may point to a lesion in this locality, the paralysis of the abducens producing strabismus, as in the cases seen by Barling, one of which had this nerve affected, while both presented horizontal nystagmus. The pupils may vary in size, the more dilated being on the side of the abscess and there may be inability to close the eyelids, with facial paralysis on the same side; this and paralysis of the oculomotor nerve may be the only localizing symptoms. Should facial paralysis occur it will probably be due to pressure of the abscess on the nerve in the pons, but as is frequently the case, a large abscess may be present and yet produce no localizing symptoms. Cerebellar ataxia and vertigo are usually due to lesions of the worm, while if the medulla be affected to a point low enough, the varied paralyses may be unilateral and on the same side as the abscess, if, however, the pons be involved more on the side of the abscess, the paralysis will be apparent on the opposite side. Macewen has well said that in all cases the symptoms of cerebellar abscess "depend upon the level of the pressure," but one may conclude that local symptoms are not usually present unless the abscess develops to the point of invading the middle lobe or the peduncle, when the so-called cerebellar ataxia develops.

The terminal stage is one of gradual death from exhaustion and coma; or sudden, depending upon the direction in which the abscess ruptures. When the pus breaks into the ventricle, the condition changes at once, the temperature rises suddenly to 103° or 105° F., the pulse becomes rapid, respirations are frequent, while there are muscular twitchings, convulsions, coma and death. When, however the abscess ruptures upon the free surface, a secondary, in-

flammatory reaction ensues leading to acute purulent leptomeningitis. The temperature becomes high and the pulse rapid and the symptoms of meningitis are fulminating, ending rapidly in the death of the patient.

The complex of symptoms, as seen in cerebellar abscess, is well exemplified in a study made by Green of four cases, in which headache as a prominent symptom was bilateral in two, frontal in one and vertical in one. No occipital headache being present in any of the cases. Paralysis of the abducens occurred in two, of which one was bilateral and one unilateral and on the opposite side from the ear disease. Optic neuritis was seen in only one case. One had general septicemia, while in the others, fever was not apparent at any time. Nystagmus on looking away from the diseased ear was present in but a single case. The abscesses were in the anterior lower portion of the cerebellum on the same side as the ear disease and in all the cases an accurate diagnosis before the operation was impossible. Leucocytosis was found in those in which the examination was made for this phase, but as the author well states, it is found in nearly 80 per cent. of uncomplicated tympanic suppurations and therefore possesses no diagnostic value in this connection.

Hammond, in five cases, based his diagnosis upon the following complex of symptoms which he regards as characteristic, being confirmed by operation or autopsy. Present or previous suppuration in the sinuses adjacent to the brain, rapid loss of flesh and strength, rapid pulse and high temperature for the first seventy-two hours, followed by a decline in the temperature and an increase in the heart action. There is also pronounced flexure of the extremities, progressive dilation without fixation of the pupils, semi-unconsciousness with uncontrollable restlessness and a peculiar indisposition to obey any request that may be made. Glycosuria, slow respiration and a tendency to go toward one side are present, while there is also a swinging of the hands toward one side, with an entire absence of paralysis.

The two groups show very well the multiplicity and confusion of symptoms, and it is only by the careful study of the individual case from all sides, that a diagnosis can be

made and the value of the symptoms even approximately estimated.

The pathologic changes in the primary bone infection consist of an area of necrosis at the point where the plate of bone separating the sigmoid sinus from the mastoid cells is extremely thin, so that erosion, as shown by Bacon, is apt to be at this point. The bone becomes discolored, softened and granulation tissue develops, while a collection of pus is found either between the sinus and bone, or behind the sinus in the direction of the occipital bone and by means of the small veins or arteries the infection is carried into the cerebellum. Or, as before mentioned, there is a direct contact between the sinus and the cerebellum, by means of the dura. This phase of the necrosis, as seen in the sigmoid groove, may be of great diagnostic value, as indicating the course of the infection toward the cerebellar region, when an exploratory operation is performed in the absence of definite localizing symptoms.

Wherever the abscess is situated, its development is usually accompanied by that of other intracranial complications, as sinus phlebitis or a small limited area of meningitis, and it is usually single, but they may be multiple and involve the cortex or deeper regions on one of both sides. Even when single, as is more frequent here than in cerebral abscesses, a smaller pus collection may be found in close conjunction with it, or an abscess here may coexist with one in the cerebrum. As the majority of abscesses in this location are chronic in character, they become capsulated by a definite lining membrane, varying in thickness from 1 to 5 mm. and although this acts as a barrier to the further spread of the infection, yet in some instances, this conservative barrier is absent, the abscess being surrounded only by a zone of softened brain substance. Whether encapsulated or not, there is always present the tendency toward greater growth and the size is extremely variable, some containing but a few drops of pus, while from others, three or four ounces may be evacuated, a case in point being seen by Woodward, in which the entire white matter of the left hemisphere of the cerebellum had broken down into an abscess cavity. The pus found in the abscess does not usually differ from that found at the origi-

nal focus of infection and it may be thick and yellow, or thin, greenish and offensive, at the same time containing particles of broken down cerebellar tissue.

The following case of cerebellar abscess was seen in consultation a short time ago and well shows the difficulty of diagnosis:

M. J., male, age fourteen years, presented the following history. The right ear had been discharging for two years following an attack of la grippe. He had been under treatment more or less constantly for the otitis, with the cessation of the discharge at times, but with its recurrence after attacks of coryza. He has lately complained of occasional dull pain over the mastoid and has become irritable, with, in addition, a considerable loss of weight. The discharge at times would become excessive in amount and the pain in the head would then become greatly aggravated. At the time I first saw him, the pain and restlessness were marked, the mastoid was not swollen, but pressure produced flinching. There was a small perforation low down in the drum, with considerable purulent discharge and a macerated appearance of the canal, but no definite areas of redness or swelling. The temperature was 98.6° F., the pulse 74 and the respiration normal. The middle ear was cleansed and drained with a small gauze wick, an ice bag was placed over the mastoid and sedatives were given internally. He only slept a few minutes at a time during that night and on the following day the symptoms had increased, although the appearance of the parts remained apparently unaltered. No other symptoms than those mentioned were present and the eye-grounds remained unchanged.

Operation was then advised and under ether anesthesia, the mastoid was opened. The cortex was exceedingly dense and no pus was found until the antrum was reached, which contained a few drops of purulent matter. Free communication was established with the tympanic cavity, a small patch of necrosis was removed, the wound was dressed in the usual manner and an improvement in his condition became noticeable in a few hours, the headache ceased and he had a good night's rest. This improvement continued for a week, when he became restless, was constantly annoyed by

trivial matters and his temperature, which had been normal, fell to 97.2° F., while the pulse was 64. I then saw him again and found him very irritable the right pupil was dilated, he complained of the light and the optic papilla was slightly swollen while he was losing flesh and strength rapidly. The mastoid wound was to all intents normal, but was redressed and when I saw him on the following day, he was semi-conscious, there was a slight stiffness of the nucha. A diagnosis of brain abscess was made, supposedly located in the cerebellum.

On account of his grave condition, it was deemed best to immediately explore the cerebellum and delay opening the mastoid wound, as it seemed perfectly healthy. A trephine opening was then made in the usual situation over the cerebellar region and when the button of bone was removed the dura was seen to be somewhat inflamed. A small incision was made in the dura and a hollow needle inserted into the lobe of the cerebellum, when, after encountering dense encapsulating wall, five drams of pus were evacuated. The abscess cavity was washed out, drained and the usual dressings applied. The patient seemed to rally well from the operation, but died twenty-four hours later from apparent asthenia. A partial autopsy only was allowed, but it showed that the sinus was involved and the infection had reached the cerebellum through the small veins, from a minute purulent and necrotic focus well back on the inner mastoid wall.

Of all the intracranial complications resulting from mastoiditis or other suppurative affections of the organ of hearing, cerebellar abscess is undoubtedly the most difficult to localize. And, as has been shown by Gradenigo, neither optic neuritis, lateral nystgmus, titubation, vertigo, nor rigidity of the nucha are special symptoms. In many cases the location of the lesions one finds in the course of a mastoid operation with symptoms of endocranial abscess such as caries of the sigmoid groove, with thrombosis of the sinus, will be the only indication that the pus collection is probably situated in the cerebellum. In the cases where a diagnosis is made in advance of operation, it is only by the careful study of the complete symptom-complex, as it is almost im-

possible to do so from focal symptoms alone, and when recognized early it is more often by a process of exclusion, rather than by any definite signs or symptoms.

The difficulties of recognition are also often enhanced by the frequent presence of complicating factors such as pyemia, sinus thrombosis or meningitis. In sinus thrombosis, rigors, high temperature, and increased heart action, are almost always present, while in leptomeningitis there is high temperature, rapid pulse and marked irritability of the special senses as predominating symptoms. Heiman believes that if due consideration be given to all the symptoms, an accurate diagnosis can be made in three-fourths of the cases, and while it is not of much value to place reliance upon single symptoms, yet in many cases one can be fairly sure of the presence of a cerebellar abscess, when certain groups of symptoms are studied in relation with one another. Cerebellar abscess may materially lower the temperature in the presence of a complicating sinus thrombosis and when a case of suspected intracranial complication is seen, the factor should be thought of. Achard and Bellanger believe that paralysis of the arm of the affected side with muscular weakness of the legs, exaggerated patellar reflex on the same side and conjugate deviation of the eyeballs toward the unaffected side, are especially indicative of this condition. And, as has been suggested by Church, in this connection, the x-rays may be of service here, the same as they have been of use in cerebellar tumors, but no cases have been recorded as yet of their value in this field.

In the differential diagnosis between temporo-sphenoidal and cerebellar abscesses, the usual error is to mistake the latter for the former, but the localizing symptoms of cerebral abscess are usually present sooner or later, and if a decision cannot be made, an exploratory operation will be of great help. Examination of the visual field may yield valuable information, but choked disc alone simply indicates an intracranial lesion and is not characteristic of an abscess in this locality and does not explain the nature of the lesion. Grant reports two interesting cases, in one the diagnosis was rendered more difficult by the presence of labyrinthine symptoms, while in the other, both ears were affected and rigors were

very prominent, due to other complicating factors. Disease of the labyrinth and semicircular canals may account for some of the symptoms as in a case reported by Bacon, with slow pulse, vertigo, vomiting, deafness, headache, etc. In this case a cerebellar abscess was supposed to be present, but the operation showed caries of the semicircular canals. In Meniere's syndrome, the headache is neither so violent, persistent, nor localized, and while in cerebellar abscess the somnolence and stupor may increase as the disease progresses this is not the case in the former.

Subnormal temperature is often present after influenza or other depressing diseases, but it is usually of short duration and not continuous like that of cerebellar abscess, while rapid emaciation may be due to the same causes, but when, in addition, the temperature is low, and there is constipation, sluggish mental action and localized pains in the head, the diagnosis is strongly in favor of the cerebellar lesion. The cerebellar gait, so-called, while of strong presumptive evidence, may be due to irritation of the auditory nerve or semicircular canals, as has been shown by Starr, and a similar syndrome be produced by defective aural drainage, without the presence of abscess at all, the symptoms being relieved after the defective drainage has been rectified. Tumors of the cerebellum, such as glioma, tuberculous, etc., may produce symptoms very similar to abscess, but the differentiation can usually be made by the presence of other factors resulting from pressure on surrounding parts.

The early stage of the abscess formation, corresponding to the initial infection from the mastoid, may last only for a day or two, or a week at the utmost, while after these acute, primary symptoms have subsided, it may remain latent for a number of years, being incited to renewed activity by the development of a new inflammation of the original focus of infection. One, and possibly two, extraordinary cases have been reported, in which the abscess spontaneously evacuated through a carious opening in the sigmoid groove, but such a result seems hardly credible and one should never, for a moment, anticipate such a termination. The usual tendency of the abscess, if not operated on, is to cause death by pressure edema of the surrounding parts, or to perforate extern-

ally with resulting meningitis; or internally, into the fourth ventricle, with immediate death from cardiac and respiratory paralysis.

While the patient will invariably die without operation, yet the success obtained in this field has been so promising that one is justified in operating as soon as the diagnosis is made, but in some cases one may wait a short time for the development of any symptoms more closely indicating the location of the abscess.

As the only treatment for cerebellar abscess is surgical, the primary focus in the mastoid should, as a rule, first be exposed and the tract of the infection traced, if possible, to its results further back. Bergman states that with a history of ear disease, persistent sleeplessness and a continuous temperature of 99° F., one is justified in opening the cranium, but the otologist may wait for localizing symptoms or until a condition of hebetude is well pronounced, before interfering. The advantages of delay are, that the location of the abscess may be more apparent latter on, while the danger to the patient is not materially increased. As pointed out by Bramwell, in cases where the diagnosis is uncertain, the operation adds but little to the risk.

It is always advisable, after cleaning out the mastoid, to expose the sigmoid sinus, while to get at the inner surface of the mastoid, the posterior portion of the bone can be removed until the dura is fully exposed for at least 2 cm. behind the lateral sinus; exploration can then be made in the cerebellum in any direction to the depth of $3\frac{1}{2}$ cm. inside of the lateral sinus. In exploring, in the absence of a precise diagnosis and in the presence of grave symptoms, it is best not to incise the dura, as the hernia which sometimes follows is a serious complication and should not be incurred, unless in the presence of immediate danger or when the diagnosis is probable. Intervention should, at times, be limited to exploratory puncture and aspiration. Gradenigo in one case by this method, aspirated fragments of cerebellar tissue and excluded a deep abscess, without in any way injuring the patient.

The general routine of procedure in these cases has been well mapped out by Pritchard, and he advises to first open

and explore the mastoid cells and antrum. Failing here to find sufficient disease to account for all the symptoms, open the cranium in the groove for the lateral sinus and so obtain access to both the middle and posterior fossa. Then examine the lateral sinus and search for both extra and intradural abscesses. Treat the sinus as indicated by any pathological changes present, and if the sinus is not thrombosed, explore the cerebral substance above and the cerebellum below the level of the horizontal groove of the lateral sinus. If there is an urgent need of operation and the diagnosis is fairly established, the brain cavity may first be opened over the supposed site of the abscess and the mastoid operation performed later. But the aural lesions should receive attention immediately thereafter, or, if the condition of the patient is such that this is impossible, the mastoid should be operated upon subsequently. But it should always be remembered that the aural lesion is the original cause of the brain disease.

When the mastoid is sclerosed or eburnated, or for any reason that will make the ordinary operation inadvisable, the skull may be opened from 5 to 7 cm. back from the edge of the osseous meatus, just below the superior curved line of the occipital bone and on a line drawn from the inferior osseous edge of the orbit to the occipital protuberance. The technic for opening the skull here is the same as in other situations, but the pus in a cerebellar abscess is apt to be so thick that it will frequently not pass through the ordinary needle, and after the dural flap has been raised, it is best to use a large grooved exploring needle. If pus be found, the cerebellum is freely incised with a probe-pointed bistoury and the abscess cavity is evacuated and a proper drain inserted.

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ABSTRACTS FROM CURRENT OTOLOGIC, RHINO-
LOGIC AND LARYNGOLOGIC LITERATURE.

I.— EAR.

**The Importance of the Surgical Treatment of Chronic
Middle Ear Suppuration.**

EDWARD BRADFORD DENCH, New York. (*Medical News*, Oct. 17, 1903.) Statistics are given which seem to show that over one per cent. of all cases of chronic suppuration will develop some severe intracranial lesion. When the radical operation for chronic middle ear suppuration is done, every vestige of diseased bone ought to be removed, nor should the surgeon stop until this is done and all sinuses, no matter how small, explored. Details of the operation are given, one point especially being insisted upon, namely: the careful inspection of the internal tympanic wall and the removal of all softened bone, which bone should be removed as thoroughly and as freely when necessary as in the neighborhood of less important structures. "It is useless to perform an operation of this kind and to leave any softened bone, although in its removal the horizontal semicircular canal may be opened or the aqueductus Fallopii may be entered and the facial nerve exposed." Vertigo, after injury of the semicircular canal, will very likely be of short duration only. The author likes to line the entire bony cavity when possible by means of Thiersch grafts, held in position by small pledgets of cotton. He has had extremely satisfactory results in these operations, the first dressing being done on the fifth or sixth day, at which time the posterior wound will be entirely healed. In some cases the ear has been practically dry in two or three weeks. Out of 70 cases operated upon by this radical procedure, 54 were cured, 11 improved and 5 were under treatment.

While the removal of the ossicula and the curettage of the tympanic walls improves many cases of chronic suppuration, it will not absolutely stop the discharge unless all diseased bone has been reached. This operation of ossiclectomy is justified in a certain proportion of cases. The surgeon should usually warn the patient that the operation may not give complete relief and that later the more radical procedure may be necessary. Where the aural discharge is persistent and profuse, the author regards the radical operation as the operation of election. *Richards.*

Tuberculosis of the Middle Ear, With the Report of a Case

Z. L. LEONARD, New York. (*Medical News*, July 4, 1903.) Tuberculosis of the middle ear, while rare, is a cause of chronic purulent inflammation. It has usually been thought to be secondary, but the writer's case would seem to be one of primary trouble, though he says infection may have come from the pharynx. It is characterized by the constant appearance of a purulent discharge without pain, followed by progressive destruction of tissue. The initial discharge may come through several small pin-hole perforations, which soon run together, forming one large circular perforation, with thick, everted edges and blueish-white edematous look. Deafness is noticed early. There is loss of bone conduction and lowering of the upper tone limit. Discharge is not usually foul in odor. Treatment is similar to that pursued in any case of purulent discharge from the ear, together with such measure as are necessary to safeguard the general health. *Richards.*

Indications for the Performance of the Mastoid Operation.

WILLIAM C. BRAISLIN, Brooklyn, N. Y. (*Medical News*, December 27, 1902.) A review of conditions under which the mastoid operation is demanded.

In acute cases, many of which will recover completely without operation, the author is inclined to be conservative, though not extremely so.

Extreme continued pain over the mastoid, with tender-

ness, varying temperature, general prostration, especially in the presence of a ruptured drum membrane, are indications for operation which should not be postponed for more than twenty-four hours. In subacute cases, where the pain is of variable character as well as the temperature, decision may be difficult, but a history of the patient, the general physical appearance and a study of the temperature chart for a few days will usually determine. Opiates ought not to be administered, as they mask the symptoms.

Richards.

The Indications for Operative Intervention in Middle Ear Suppuration.

BARTON H. POTTS, Philadelphia. (*American Journal of the Medical Sciences*, July, 1903.) Profuse, long-continued discharge is suggestive, but not diagnostic of antrum or mastoid involvement. Tenderness over the mastoid, an inflamed, tender, bulging upper and posterior canal wall, and a sinus, whatever its location, leading into the mastoid are the most positive indications for operation. In cases of pyemia, rapid fluctuations of temperature through a range of several degrees is the most important sign. The slow, thready pulse, out of proportion to the elevation of temperature, with irregular or sluggish pupils, rapid failure of health, rigors or convulsions are signs of intracranial pressure which should demand immediate operation.

Richards.

The Prognosis and Treatment of Chronic Deafness.

PHILIP D. KERRISON, New York. (*Medical Record*, November 21, 1903.) Ninety-five per cent. of all cases of deafness are due primarily to tympanic disease or to disease in some part of the conducting apparatus. Chronic tubal catarrh in which there is marked narrowing of the Eustachian tubes, and chronic hypertrophic catarrh of the middle ear, in which the tympanic mucosa and all the joint structures of the ossicular chain may be involved, is very much improved by the re-establishment of the Eustachian tube to

to its normal condition and caliber and the restoration of the tympanic membrane as nearly as possible to a healthy condition. To bring this about, inflation by catheter at regular intervals; the application of astringent solutions to the nasopharynx and pharyngeal mouth of the tube; medication of the tubal mucosa by stimulating vapors; mechanical dilation by graduated bougies, or by electrolysis are all of great value; the patency of the tube once obtained, being maintained by occasional gentle inflation, together with pneumatic massage of the membrani tympani in order to exercise the ossicles and restore their mobility.

Deafness due to suppurative processes in the middle ear is usually less than would be expected considering the apparent damage to the conducting apparatus. Most patients of this class apply for treatment, not on account of deafness but for the relief of a chronic purulent discharge. When they seek relief on account of the hearing, the stapes may be found buried beneath the mass of cicatricial tissue, and the logical treatment would be the division and removal of this tissue. When this is done there is often an immediate brilliant result, but as the operation wound heals the initial gain is almost invariably lost, though the final net result may show an appreciable improvement.

Chronic hyperplastic otitis media or dry catarrh is the most discouraging form of deafness which the otologist has to treat. It is apparently a productive inflammation from the start, in which the newly formed connective tissue contracts, pressing upon the normal structures of the tympanic mucosa, which is converted into a dry, sclerotic membrane, closely adherent to the tympanic walls. The same changes are often found in the mucous lining of the Eustachian tube, the passage of which may be abnormally wide, while the drum membrane is usually not retracted. The deafness is due to impaired mobility of the ossicular chain, and the ankylosis is very apt to be a true one. Although the drum membrane may be normally placed and free, the ossicles are held by constricting bands which bind even the stapes to the walls of the oval niche and window. In the initial stages some beneficial results may possibly occur from the use of stimulating vapors thrown into the middle ear

cavity through the catheter, together with forcible inflation at long intervals. Surgical aid has been proposed to remove under the most rigid aseptic precautions the drums, malleus, and incus, great care being observed to separate the incus from the stapes without injuring the latter or dislodging it from its position within the oval window. The writer himself has never felt justified in advocating this operation, nor have the results of such cases as have been operated upon seemed altogether favorable, although the immediate result of the operation is probably almost always a decided improvement in hearing. The after-danger lies in the tendency of adhesions to re-form between the stapes and adjacent structures, the prevention of which is very difficult if not impossible. *Richards.*

Carbonic Acid Apparatus for Inflation of Eustachian Tube.

LUCAE. (*Int. Centralbl. f. Ohrenheilkunde*, No. 6. Bd I.) The author has designed an apparatus for the inflation of the Eustachian tube by means of compressed carbonic acid. The bottles filled with 5 kilos of fluid carbonic acid is connected with a manometer which regulates the pressure. The author has used the apparatus very often and has never seen untoward results from its use. The reddening of the promontory seems a little greater when carbonic acid is used than when ordinary air is used.

Anomaly in the Position of the Sinus.

BITTER. (*Int. Centralbl. f. Ohrenheilk.*, No. 6. Bd I.) The operation was undertaken on account of an acute otitis media. The sinus lay so far forward that it touched the posterior meatal wall, so that a part of this had to be removed, and a large part of the sinus was laid bare in order to reach the antrum. After the operation, the patient experienced very severe pain. On changing the bandage, the free lying sinus wall showed itself to be very sensitive to the lightest touch. Orthoform was dusted on the wound with the result that the pain disappeared as soon as its effects commenced, to reappear two days later in a milder form, as soon as the

anesthetic properties grew less. The same observation was made at every change of dressing until the sinus wall was fully granulated over.

Bony Changes Following Chronic Middle Ear Suppuration.

BITTER. (*Int. Centralbl. f. Ohrenheilkunde*, No. 6, Bd I.) The author had performed a radical operation on account of chronic suppuration of the middle ear. The mastoid was sclerosed, and the antrum small; a small fistula led from the antrum into the middle ear. The entire region of the aditus behind this fistula was changed into a sclerosed mass of bone from which a part was chiseled off in order to obtain a view of the cavity. The contour of the semicircular canal was not seen, and no lumen was obtained by the chiseling. The remnants of the hammer were extracted, but the anvil could not be found, and apparently had been incorporated into the mass of bone. After the operation, the wound behind the ear was closed primarily by a Koerner's flap. This became necrotic, and the epidermising of the cavity proceeded slowly. Thiersch's flaps were transplanted, and epidermising followed rapidly. Suddenly most of the newly formed epidermis became necrotic, and it was impossible to prevent its loss. This phenomenon occurred simultaneously with the great cold in November, 1902, so that the author thought of a relationship between these two. The patient was compelled to wear the bandage which had been removed after the closure of the retroauricular wound, and the healing progressed undisturbed.

Otogenous Pyemia, With Pachymeningitis Interna Circumscripta Acuta.

ALEXANDER. (*Int. Centralblat. f. Ohrenheilk.*, No. 7, Bd. I.) The case, where discharge from both ears had existed since early childhood, belongs to the group of the pulmonary forms of otogenous pyemia. When the patient was received, there was already abscess of the left lung and weakness of the heart. The right ear was the starting point for the pyemic symptoms, and at the radical operation a

cholesteatoma of almost the size of a walnut was removed from it; there was also found a fetid thrombus adhering to the wall of the right lateral sinus. As the local pulmonary symptoms increased, and the abscess became bilateral, exitus lethalis was the result. Post mortem showed, among other things, an acute pachymeningitis interna corresponding to the convexity of the right cerebral hemisphere. Arachnoidea and pia as well as the surface of the brain were entirely normal. The case usually result in a much greater involvement of the dura. The author's case shows a very early stage of a pachymeningitis interna, such as are seldom observed anatomically, because in the meningeal forms of otogenous pyemia, which these cases usually are, the exitus lethalis usually occurs in a stage where there is an extensive purulent involvement of all of the meninges, and occasionally of the brain. In operations at such a stage pachymeningitis would oftener be seen if we were accustomed to open the intradural space in cases of extensive pachymeningitis externa or purulent change on the mesial wall of the sinus. An opening under these circumstances is equivalent to opening the peritoneal cavity. The author did not ligate the jugular because (1) he had demonstrated metastasis in the lungs, and (2) because the narcosis and operation had to be shortened on account of the weakness of patient's heart.

Keloids of the Lobule.

ALEXANDER. (*Int. Centralbl. f. Ohrenheilk.*, Bd. I. No. 6.) (1) The summary of facts collected by Alexander are as follows: The keloids of the lobule do not show a periarterial arrangement of the fibres. The specimens give no support to Warren's theory of the origin of the keloids by proliferation of the arterial walls. (2) According to the histologic appearances, the growth of the keloid takes place from the normal connective tissue of the corium, which, when hypertrophied, compresses either partially or totally the regional blood vessels and causes their disappearance. (3) The characteristic tumor cells in the arterial walls described by Warren were not found in the tumors examined. (4) In respect to the etiology, the cause of the arising

of keloid of the lobule seems to be the act of piercing the ears, or perhaps the subsequent purulent inflammation of the canal formed and its vicinity, coupled with a predisposition of the patient toward keloid formation. (5) According to experiences on other parts of the body and the reaction of the skin involved, the wearing of ear rings, or perhaps their weight, cannot be regarded as the only cause of keloids. We must assume that a scarcely perceptible new-formation of connective tissue is found at some point in the vicinity of the hole in the lobule (which is caused by inflammation following the piercing of the ear) and this, years later, when the individual's constitution is changed, gives origin to keloids.

Anatomical Findings in Adhesive Processes in the Middle Ear, and their Relation to the Diagnostic Appearance on the Drum.

POLITZER. (*Int. Centralbl. f. Ohrenheilk.*, Bd. I, Nt. 7.) The author bases his conclusions on a large number of cases examined intra vitam, and post mortem, and an observation of the disease extending over years. The newly formed adhesive connective tissue acquires a greater extent and strength in the purulent processes in the middle ear, then in the non-purulent, catarrhal conditions. Still, Politzer has seen in the latter strong bands and bridges which bound the drum to the inner wall of the tympanic cavity. The adhesions caused by the formation of connective tissue affect on the one hand larger or smaller parts of the drum, and on the other the inner wall of the tympanic cavity or the anvil and stapes, or the aural bones lying in the attic and the bony wall of the attic. Politzer distinguishes the surface approximation of the drum to the promontorial wall and abnormal union of the drum and ossecles with the walls of the tympanic cavity by means of thickening bands and membrane. The latter, Politzer considers to be in the majority of cases thickened and hypertrophic folds of mucous membranes which are found in varying amounts, in the tympanic cavity and antrum mastoideum as remnants of embryonal mucous membrane folds. Politzer bases this assertion

on the fact that in all the cases examined anatomically by him the presence of such bridges in the middle ear was connected with just such stellate, branching and thickened connective tissue membrane. Politzer sketches among a large number of very instructive pictures of findings on the drum, the otoscopic appearance of the adhesions in the neighborhood of the drum, and especially emphasizes the diagnostic importance of the changed appearance of the drum when examined with Seigle's speculum, and its importance for the often very fortunate intratympanic operations.

Circumscribed Gangrene of the Cerebellar Dura, Following Chronic Suppuration of the Middle Ear.

HEINE. (*Int. Centralblatt, f. Ohrenheilkunde*, Bd. I, No. 6.) The patient, aged 20 years, who had suffered from childhood with a foul smelling discharge, gave the following appearance on presenting himself: The right mastoid was very sensitive, foul pus was discharging from the meatus; almost total defect of the drum. Conversational tones heard at the ear. The fundi of the eyes showed venous hyperemia, the disc distinct. Severe headaches and torticollis. Sensorium clear. Temperature 38.3 C. Immediate operation. With the first cuts of the chisel, pus and granulations were revealed. The bone in the region of the sinus was greyish yellow discolored, but the sinus looked healthy. Puncture revealed normal appearing blood. The cerebellar dura was discolored for the space of the size of a quarter. Repeated punctures gave no pus. Eight days after the operation, pus bubbled out of a small fistula on pressing on the dura. The bone was removed down to the vertical semicircular canal and a small abscess opened in the dura. On changing the dressings pus still exuded from the depths of the wound; 5 days later, on changing the dressings a piece of the dura, size of a nickel, and a smaller gangrenous piece were removed, whereupon a large amount of cerebro-spinal fluid was discharged, which continued for weeks, gradually growing smaller in amount. The patient acquired tuberculosis of the lungs while in the hospital, which increased in severity and caused his death in six months. As the author learned

of his death too late, he could not make a special post-mortem, and in the regular report of the post-mortem, nothing of importance was found. When patient was first seen the diagnosis of a diffuse purulent leptomeningitis was made. In another case, the lumbar puncture was made which gave purulent cerebrospinal fluid and so confirmed the diagnosis. In this case, only after the removal of the gangrenous part did it become clear that it was a case of circumscribed meningeal gangrene. In the statistics of Koerner, 6 cases of circumscribed gangrene of the dura are reported, 4 from Macewen, 1 from Lucae, and 1 from Somsen, all of which, however, affected the middle cranial fossa, while in this case the cerebellar dura was gangrenous.

Two Cases of Aneurism of the Arteria Carotis Cerebri.

ZUR-MÜHLEN, Riga. (*Archives of Otolaryngology*, Vol. XXXII, No. 5.) CASE 1. A woman, aged 57, with a probable syphilitic history, fell heavily on the right side of her head; for an hour she was totally unconscious and vomited profusely throughout the day. For a time the sense of smell was lost on one side and tinnitus in the right ear has persisted since the fall. Inclining the head to the right and pressing the carotid of that side, lessens and sometimes suppresses the tinnitus, with a stethoscope a loud systolic murmur of uniform intensity could be heard over the entire head; if the right common carotid be compressed the murmur is decidedly weaker. Although the location of the aneurism is to a degree conjectural, yet, because of the temporary anosmia evidently due to pressure on the olfactory bulb, it is probably located at the point where the carotid branches off into the anterior and middle cerebral arteries.

CASE 2. A woman aged 24, when 2 years of age, struck her forehead against the sharp edge of a marble table. When 4 years old, she became unconscious without any apparent cause and remained so for three hours. There were no convulsions. At the age of 10 severe headaches began, at first daily intermittent, later, continuous for weeks and months. For the last 10 years, exophthalmos and dilatation of the veins around the eyes have been well marked.

Upon both sides of the occiput at the place of exit of the mastoid veins one can feel a marked fremitus, diffused backward, upward and downward. With the stethoscope one hears a loud systolic murmur over the entire head. If the right carotid is compressed the murmur becomes less and the palpable fremitus at the right occiput has gone. Simultaneous compression of the left carotid caused complete disappearance of murmur and fremitus. There was bilateral, pulsating exophthalmos, a left hemianopsia, and atrophy of the corresponding halves of both optic nerves, and a hemianopic reaction of the pupils. In the right ear the lower tones were poorly heard, C heard only when the fork was struck very hard. In this ear subjective tinnitus of a severe beating and knocking.

The diagnosis was aneurism or rupture of the right carotid into the cavernous sinus.

Under cocaine anesthesia the common carotid was tied high up in the neck. The eyes gradually receded, the fremitus disappeared, while the murmur was much less perceptible. The severe headaches disappeared, sleep is more normal and there is less tendency to somnolence. The hearing is improved and there is great diminution of the subject-sounds.
Campbell.

On the Pathological Anatomy of Deaf-Mutism.

SCHWABACH, Berlin. (*Archives of Otology*, Vol. XXXII, No. 5.) In a patient who died of general miliary tuberculosis the author obtained possession of the right temporal bone only. The middle ear presented evidences of the implantation of recent tuberculosis on an old purulent otitis media. In the internal ear the main changes were found in the middle part of the basal turn of the cochlea, where they obliterated the cavity, the peri, as well as the endo, lymphatic spaces, so that nothing was to be seen, of the membranous structures. In the basal turn the auditory nerve and its terminals were diminutive or entirely absent. The aquaeductus cochleae could not be recognized. The aquaeductus vestibuli was clearly seen and presented no changes. Toward the anterior part of the basal turn, the bony, as well

as the connective tissue, new formation diminished gradually and were completely absent in the upper turn.

Campbell.

On Hemorrhage from Arrosion of the Brain Sinuses in Suppuration of the Temporal Bones.

EULENSTEIN, Frankfort-on-Main. (*Archives of Otolaryngology*, Vol. XXXII, No. 5.) A child aged 5, developed mastoiditis during an attack of scarlet fever. In spite of free paracentesis the tenderness and swelling about the mastoid increased.

Operation disclosed openings leading to the middle and the posterior cranial fossae. The sigmoid sinus showed respiratory motions but its middle portion was covered with granulations and had about it a large extra-dural abscess. Ten days later the sinus wall gave way, but the profuse bleeding was controlled by compression bandages. During the course of the next 4 days, by retention of the secretions, through fear of hemorrhage in changing dressings, the temperature rose to 40.5 C. and a violent chill set in.

A change of dressings was imperative. On attempting to lift the external plug a new hemorrhage occurred but stopped immediately on replacing the tampon. The internal jugular and facial veins were tied in two places and severed and then the lateral sinus was exposed between the knee and the torcular.

While pressure was made on the sinus the dressings were changed without much loss of blood. The general condition from this time on steadily improved.

In reviewing the literature the author finds, including his own, 18 cases on record; of these 13 occurred in chronic suppurations, 4 in acute and 1 no statement. *Campbell.*

General Sepsis in Chronic Suppuration of the Middle Ear, with a Central Perforation of the Drum.

BEZOLD, Munich. (*Archives of Otolaryngology*, Vol. XXXII, No. 5.) The three cases reported occurred in healthy individuals, complicating a form of middle ear inflammation

which experience has taught us to look upon as harmless and thus led to the assumption that the infecting element was very virulent or that it was present in exceptionally large quantity. The predominating, micro-organism was in each case the streptococcus pyogenes. In two of the cases there was additionally an otitis externa crouposa and in one of them a furunculosis. The first case was complicated by a tonsillar exudate and in the last on opening the mastoid several dark red glands were cut across. This makes it seem that there was a simultaneous infection of the middle ear mucous membrane and the lymphatic system.

Campbell.

Recent Theories on Sound Conduction.

TREITEL, Berlin. (*Archives of Otology*, Vol. XXXII, No. 5.) After reviewing recent literature, the author finds that more extended research is needed to settle the question. He thinks that it does not detract from Helmholtz's credit if his theory has been amplified or modified. All agree that for high tones, conduction through the ossicles is of little importance. Conduction through the ossicles is of little importance. Conduction through Mt. and the chain of ossicles is still accepted for low tones. As to the middle ear Beckman sees only a dampening apparatus, for movements of the very unstable labyrinthine fluid.

Rinne's experiment is of undoubted value in the diagnosis of ear disease. There is still a doubt as to how sound waves excite the fibres of the basilar membrane; bone conduction is sufficient in itself to produce this, as is seen in cases of loss of Mt. and in fixation of the stapes with lengthened bone conduction.

Campbell.

Ankylosis of the Stapes.

DENKER. (*Klinisch-Therapeut. Wchnsch.*, Vol. 10, No. 30.) In all cases of bony stapes fixation, there existed a transformation of the normal bony tissue of the stapes, and of the neighboring bone of the niche of the oval window, in which osteoid tissue, and later spongy tissue were formed as a result of which the annular ligament in some cases, have entirely dis-

appeared, in the newly formed bony masses, whereas in other cases the stapes was united with the margin of the window by bony strands.

With regard to the etiology of the affection, the writer believes that it is not only theoretically possible for a middle ear affection to lead to an ossifying periostitis, and produce such changes in the bone, but it is even extremely probable, in view of the frequent association of the two affections, that a casual relationship exists, at least for many cases.

This assumption does not, however, explain the method of origin of the spongy tissue in all the cases, which show no alteration in the mucuous membrane of the tympanum, which could be considered as a result of previous inflammatory processes.

In such cases, we must assume a primary involvement of the periosteum, or of the bone. It is established that the spongy alteration may take place without involvement of the periosteum, but as a rule the periosteum of the tympanum or of the vestibule is involved.

The question of the etiology of the capsule of the labyrinth is partially answered by statistic which show that the bony ankylosis of the stapes occurs, especially in women, who attribute in most instances, their affection to pregnancy or childbirth. In many other instances, however, constitutional anomalies must be considered, especially in view of the fact that the affection begins usually on both sides, and with the same degree of intensity.

The diagnosis the stapes ankylosis, is not difficult. If the tube is open, and the drum membrane normal, we may assume that stapes fixation is present, when the functional test shows marked diminution of hearing, and negative result with Rinne's test. Cases in which the diagnosis of stapes ankylosis was made on these grounds, have, without exception, at autopsy, shown the correctness of the diagnosis.

With regard to treatment, the writer believes that all local procedures, ranging from simple Politzer inflation to an extraction of the stapes, have given unsatisfactory results, that they are not to be recommended, especially since in many cases, an actual injury may possibly result. Although it is not possible to improve the hearing by therapeutic measures,

in many instances, however, the subjective sounds may be favorably influenced by massage of the drum membrane.

Goodale.

Case of Spontaneous Dislocation of the Incus, with Fistulous Rupture into the Bony Meatus.

IMHOFFER. (*Prager Med. Wochen.*, Sept. 3, 1903.) A child two and a half years old was brought, showing marked emaciation, following gastro-enteritis, pertussis, general furunculosis, and multiple glandular enlargement. The right meatus showed abundant pus, while the drum membrane appeared reddened, swelled and covered posteriorly with granulations. Treatment was carried out for three months with improvement, when fever suddenly set in, and inspection showed the region behind the left ear markedly swollen and reddened, the mastoid process sensitive, while the posterior, upper wall of the meatus was pushed forward. Operation was on the point of being performed, when the mother of the child refused to give her consent. Two days later, the severe symptoms improved, the temperature fell to normal, and the swelling subsided. Two months later, the mother noticed a body in the ear which prevented syringing and cleansing. Examination showed a mass extending into the lumen of the external meatus, and closing it almost completely. The pea-sized mass was removed, and proved to be the incus, imbedded in granulations. Examinations subsequently showed a fistula at the spot where the mass was imbedded, which extended in the direction of the mastoid antrum. Subsequently a sequestrum of bone was thrown off, which came apparently from the vicinity of the antrum. The child continued to improve, and is now in excellent condition.

Goodale.

II.—NOSE AND NASO-PHARYNX.

The Etiology of Nasal Polypi.

FRANCIS R. PACKARD. (*American Journal of the Med-*

ical Sciences, November, 1903.) Packard thinks that in most cases polypi have their origin in diseased tissue, especially in some part of a necrosed ethmoid; that they are not mere tumors, the removal of which will cure the condition, but that they are results of pathological conditions, and that they should be carefully studied with reference to the underlying conditions which are causative and the correction of these attended to. *Richards.*

The Diagnosis and Treatment of Inflammation of the Accessory Nasal Sinuses.

JOSEPH S. GIBB, Philadelphia. (*American Medicine*, July 25, 1903.) Exploration of the maxillary antrum with trocar and canula and the syringing out of the cavity with pus is recommended as a diagnostic measure, with which procedure the reviewer is in entire accord, having found it to be almost painless for the patient, done with comparative ease, and to give immediate positive information where one might otherwise be a long time in doubt as to the diagnosis. Treatment is entirely surgical, the author regarding it as a waste of time to attempt to wash out this sinus through the natural opening. He considers perforation of the canine fossa the preferable route to reach the sinus, as a larger opening can thereby be secured, and no sound teeth are removed. A sufficiently large opening should be made so as to allow for the free use of curettes and other instruments. A good sized opening being made in the canine fossa, the after treatment consists in packing the sinus with antiseptic gauze or introducing a good sized drainage tube. Irrigation is continued until all evidences of suppuration disappear. Considerable time is sometimes required before this is brought about.

In cases of frontal sinus suppuration he rather leans toward the open method when external operation is performed, inasmuch as the latter allows the sinus to be kept open for an indefinite time, with later possible cicatrization and obliteration of the sinus. The incision is made along the superciliary ridge down to the bone; a button of bone removed over the sinus and the latter thoroughly cureted. Reference

is also made to inflammation of the ethmoid and sphenoid cavities.

Richards.

Nasal Polypi: A Study of One Hundred and Forty-seven Cases.

J PAYSON CLARK, BOSTON. (*Boston Medical and Surgical Journal*, July 2, 1903.) Histologically nasal polypi must be considered as stretched and edematous mucous membrane, not as true myxomatous tissue. The author found no evidence that constitutional diathesis or impairment of general health stood in any causative relation to the development of polypi, nor does he think that the septal deformity or nasal obstructions have much of any relation except that a deformed septum might bring about a condition favorable to the growth of polyps. A neglected injury to the nose, with its attendant nasal obstruction, impairment of the circulation by pressure, and irritating muco-purulent or purulent discharge from the injured portion, might quite conceivably result in the formation of edematous granulations which would eventually develop into full-fledged polyps.

With reference to ethmoid disease, he does not think that all cases are due to that as a cause. Four-fifths of his cases gave a history of frequent head-colds, but he thinks that in many cases these colds are only symptoms of existing polypi. Sneezing was marked in one-third of the cases. Thirty-nine out of ninety-three had lost the sense of smell; four were much impaired; twenty-seven impaired; leaving twenty-seven, or somewhat less than one-fourth in whom the sense of smell was presumably normal. Bronchitis was noted in 21 cases and asthma in 10; asthma and bronchitis in 14; hay fever in 4. No case in which the nasal polyp took on a malignant character was observed. In removal the galvanocautery was not used, and he is opposed to its use, as it may tend to excite the condition in the mucous membrane which we are trying to get rid of. The middle turbinate will frequently have to be removed, as it is sometimes so deeply affected by the process that only its thorough removal will suffice to eradicate the disease. In some cases the application of 95 per cent. alcohol on pledgets of cotton to the site of the growths seemed to be of some assistance in preventing

the recurrence of polypi. Only a small portion of cases are caused by sinus disease. A cure will usually result if patients will return sufficiently often for treatment. *Richards.*

Cheesy Empyema of the Nasal Accessory Sinuses.

STIEDA, Königsberg. (*Archives of Otolaryngology*, Vol. XXXII, No. 5.) The author reports three cases in which the pronounced feature common to all was the extensive formation of fetid cheesy material in the nose. This was easily removed with a dull curette. In all three cases the fistula formed a communication with the surface in the orbital region. In the one case was found in almost pure culture the bacillus coli communis, in two an amorphous material, while the material removed from the third contained connective-tissue cells, many of them broken down, looking like cut sections of the acini of glands.

The cases were readily cured by the establishment of free drainage and keeping the cavities cleansed. *Campbell.*

On the Influence of the Enlargement of Portions of the Pharyngeal Lymphatic Ring in General, and Adenoid Vegetation of the Naso-pharynx in Particular, upon the General Well-being of the Organism.

W. N. NIKITIN. (*St. Petersburg Med. Wochen.*, July 5th, 1903.) The author considers a normal tonsil, whether a lingual, pharyngeal or faucial, to be one which is not elevated above the surrounding surface. He therefore agrees with Bosworth in considering the tonsils of the anatomist as pathologica structures. In the majority of persons, with enlarged tonsils one finds also enlargement of other portions of the lymphatic ring. In all such swellings, one can distinguish two types, namely, those with a small amount of connective tissue, and others which are firmer, and show connective tissue strands. As time goes on, the latter begin to predominate, and the follicles, as a result of cheesy or calcareous degeneration, or by cicatricial changes, disappear, and the connective tissue bundles become cirrhotic. The author does not regard these manifestations as evidence of natural healing.

any more than in the case of sclerosis occurring in an enlarged liver. The symptoms of mechanical disturbance become less and less with time, but secondary alterations persist, and excite later new and peculiar disturbances.

After reviewing the symptoms due to enlargement of these structures, the author proceeds to discuss the question of infection in the different regions.

It is interesting to note that late forms of syphilis and hereditary syphilis excite a hypertrophy of the lingual tonsil rather than an atrophy. The bacilli of tuberculosis make their entrance into the system, particularly through the pharyngeal and faucial tonsils. In tuberculosis of the pharyngeal tonsil in children, there is always the possibility of an extension, directly through the lymph channels, into the base of the brain, producing basilar meningitis.

The author believes in the complete removal of the pharyngeal and faucial tonsils, but does not employ an anesthetic for the purpose

Goodale.

On the Relation of Oral Diseases to those of the Nose and Naso-Pharynx.

KOSTLIJANETZ. (*St. Petersburg Med. Wochen.*, April 5, 1903.) In 1,600 patients examined with alterations in the nose or naso pharynx, 107 showed otitis (acute 34, chronic 53, healed 20), 167 showed Eustachian catarrh (acute 41, chronic 120), 46 showed sclerosis, 4 showed deafness as a result of intoxication, 11 showed affections of the labyrinth, 3.29 per cent. in all. The alterations in the nose or naso-pharynx were either directly or indirectly produced by continued early inflammatory affections. The author regards this percentage as perhaps actually too small, and believes that the number of ear diseases owing their origin to the nose or naso-pharynx is in reality larger.

Goodale.

On the Relation Between So-Called Adenoid Vegetations and Enuresis Nocturna.

ZWILLINGER. (*Medizinisch-Chirurgische Presse.*, Oct. 3, 1903.) The author found in 113 cases of adenoids, six

children who showed enuresis nocturna. Three of the cases were immediately cured after the operation. In all cases, therefore, of this affection the author recommends the examination of the child for adenoid vegetations. *Goodale.*

III.—MOUTH AND PHARYNX.

Tuberculosis of the Tonsils and the Tonsils as a Portal of Tubercular Infection.

HENRY KOPLINK, New York. (*American Journal of the Medical Sciences*, November., 1903.) As compared with the pyogenic forms of infection, the tonsil is the seat of primary tubercular infection but rarely. The tubercular tissue is found in the form of giant cells and tubercular nodules. It is exceptional that tubercle bacilli are abundant. Ulcers are rare. The lymph nodes leading from the tonsil are usually affected. Tonsillar tuberculosis may be the source of a general tuberculosis. So far as the cervical lymph nodes are concerned, if the seat of isolated tubercular infection of a primary nature, it can be justly claimed that such infection has proceeded from the tonsil. *Richards.*

A Case of Lipoma of the Tonsil.

CLEMENT F. THEISEN, Albany. (*Albany Medical Annals*, August, 1903.) Cases of lipoma of the tonsil are rare, the author being able to find but six recorded cases. All of these are since the year 1893. The case reported was in a girl of eight years who had had a troublesome cough more or less of the time since three years of age. The tumor was first discovered at that time and an operation advised, but for some reason was not done and the child was not seen again for five years. The tumor, which had grown somewhat larger, was attached to the center of the right tonsil by a rather long, thin pedicle coming from a tonsillar crypt almost in the center of the tonsil. It was removed by cutting it off as close to the tonsil as possible. Histologically the specimen consists of a

small, distinctly pedunculated tumor, globular in shape, measuring 7 mm. in diameter. The pedicle was 3 mm. in length. The outer surface of the tumor was covered by a thin, gray membrane, resembling skin in appearance. The greater portion of the tumor consisted of typical areolar tissue, containing vessels quite uniform in size and normal in character.

Richards.

The Causation and Treatment of Postnasal Discharge.

PERRY G. GOLDSMITH, Bellville, Ontario. (*American Medicine*, October 3, 1903.) The author has found chronic inflammation of the pharyngeal mucous membrane to be frequently accompanied by gastro-intestinal trouble and sometimes by rheumatism.

The vault is thoroughly cleansed with an alkaline post-nasal douche and then pigments of silver nitrate, iodine and carbolic acid, argyrol and protargol are used, as circumstances may seem to indicate. Scraping the naso-pharynx with a dull curet will occasionally give good results. Cases with dry, glazed condition of mucous membrane are especially obstinate and are usually associated with a similar condition of the nasal mucous membrane. Amelioration of the symptoms is the best that can be promised. There are certain cases of naso-pharyngitis of the neurotic type where almost nothing is found on examination but where much is complained of. These cases occur mostly in women whose nervous stability is below par. Measures directed to improving the general condition are indicated and little or nothing should be done by way of local applications. Imaginary operative measures sometimes produce great benefit, especially when accompanied by positive assurances that there is nothing seriously wrong.

Richards.

Hemorrhage Following Tonsillotomy, with Report of a Serious Case.

ADOLPH H. URBAN, Buffalo, N. Y. (*American Medicine*, July 4, 1903.) Patient was a boy of 7 who had been operated on at 2 p. m. of the day previous for hypertrophy of

each tonsil. No serious hemorrhage occurred immediately after, nor had any occurred as late as six hours after when he was seen by the physician who performed the operation. Sometime during the following morning hemorrhage commenced, and when seen at 5 p. m., of the day following the operation, consciousness was still present but he was unable to articulate. There was considerable vomiting of dark colored blood but no expectoration or bleeding from the mouth. A parenchymatous spurting hemorrhage was seen in the left tonsillar region, the blood being swallowed. Chloroform was given and a Paquelin cautery, heated to a dull cherry red color, applied directly to the bleeding surface. Hemorrhage ceased immediately, 700 cc. of normal salt solution was injected into the left median basilic vein. Recovery was uneventful. This case is interesting in that it occurred in a child, where serious hemorrhage after tonsillotomy is not usually expected, and that it occurred 12 to 18 hours after the operation. It was probably due to either an anomalous tonsillar or internal maxillary artery. *Richards.*

The Complications of Hypertrophy of the Pharyngeal Tonsil.

GEORGE B. WOOD, Philadelphia. (*American Medicine*, October 3, 1903.) While admitting that the tonsils have certain good physiologic properties, the amount of harm which hypertrophy of them is likely to bring about is much greater than any possible good, since hypertrophy gives greater chances for absorption of poison, while the increased manufacture of lymphoid cells will in some cases bring about some pathologic condition of the blood. More serious complications due to hypertrophy of the pharyngeal tonsil are otitic disturbances. The child with adenoids is more often taken to the physician on account of symptoms referable to the ear than for any other reason, and the large majority of cases of ear diseases in children are directly due to hypertrophy of the pharyngeal tonsil. The condition of this organ should always be investigated in cases of ear disease in children. In all cases of hypertrophy the pharyngeal tonsil is likely to become a constant source of infection to the whole of the respiratory tract. *Richards.*

The Lymphatic System and the Tonsils.

HENRY L. SWAIN, New Haven, Conn. (*The American Journal of the Medical Sciences*, July, 1903.)

Acute General Infections Originating in the Lymphoid Tissue of the Upper Respiratory Tract.

HENRY L. SWAIN, New Haven, Conn. ((*The Philadelphia Medical Journal*, December 13, 1902.) The author reports a case where tonsillar enlargement was dependent upon general lymphadenitis. The faucial tonsils enlarged enormously from time to time as did the lingual and pharyngeal tonsils, the attacks occurring usually in connection with the general lymphadenitis. The patient was 63 years old when first seen and had been subject all his life to such changes. He was under observation two years. The most benefit was obtained from Fowler's solution. He finally died from exhaustion. The tonsils enlarged so that they met in the middle line. No operation was performed on them, the treatment being mostly the use of Fowler's solution, the most benefit being obtained from this. The blood count sometimes showed an increase in the white cells; at others it was nearly normal. The author regards the tonsils, both pharyngeal and faucial, as a part of the general lymphatic system and to be treated accordingly, and does not think one's whole duty to a patient suffering from these conditions is done when the operation of ablation of a portion of the faucial tonsil or even the whole of it has been performed. Further studies ought to be given to the subject of the relation of the tonsils to the general body economy.

The pharyngeal tonsil is liable to acute attacks of inflammation in much the same way as is the faucial tonsil and much oftener than is generally considered. The typical symptoms of acute inflammation of the faucial tonsil are given, and several cases illustrating the same, cited. In these cases blood examinations show but little. In many cases when the diagnosis of inflammation of the pharyngeal tonsil is in doubt, shrinking of the tissues of the nose with cocaine and suprarenal will give positive information. In

many children post-nasal examination with the mirror is not impossible if sufficient care is taken. While digital examination is, of course, always possible, the information which it gives is limited somewhat to the question or not of enlargement, while the character of the inflammation and the condition of the surface cannot be determined with anywhere near the accuracy which is possible by examination with the mirror or indirectly through the nose.

Richards.

IV.—LARYNX.

Hypertrophic Tuberculosis of the Larynx.

CLEMENT F. THEISEN, Albany. (*American Journal of the Medical Sciences*, November, 1903.) This is a form of tuberculosis in which hypertrophy of all the structures of the larynx but no ulceration takes place. The case reported was a man of 40 years, weight 175 pounds, nearly complete aphonia, losing flesh, lungs negative. Tubercle bacilli had been found in the sputum in a former examination, but were not evident when first seen by the writer. The mucosa of the larynx was reddened, with well-marked thickening in the interarytenoid space, which was firm to the touch. Both arytenoids were thickened and infiltrated, and both cords. There were circumscribed areas on both ventricular bands, with broad bases, presenting the appearance of hyperplasia of the tissues, and covered with perfectly intact, smooth mucous membrane of grayish red color. There was no ulceration anywhere, nor was the epiglottis involved. A diagnosis of laryngeal tuberculosis was made and patient sent away. He was seen by other physicians and a diagnosis of pachydermia laryngis was given. Subsequent history with death from general and well-marked laryngeal and pulmonary tuberculosis at Saranac Lake proved the diagnosis of tuberculosis of the larynx. The literature of the recorded cases is given at some length.

Richards.

The Early Manifestations of Laryngeal Tuberculosis; Their Frequency and Treatment.

H. H. BRIGGS. Asheville, N. C. (*Jour. Amer. Med. Asso.*, December 19, 1903.) Most subjects of pulmonary tuberculosis show in the larynx, prior to the characteristic tumefactions and infiltrations, either a uniform thickening of the mucosa or an irregular thickening and hyperplasia of reddish hue of the ventricular bands and arytenoid commissure; presenting dilated blood vessels and covered with thick grayish mucus, at the start not differing materially from the laryngitis of diathetic or climatic cause. The acute form of laryngeal tuberculosis usually begins after softening has taken place in the lung and when the laryngitis has reached its crisis. The first sign is hyperemia, following which are multiple erosions on the laryngeal surface of the epiglottis and arytenoids, soon to be followed by tumefaction in the epiglottis or aryepiglottic folds. The cough is hacking with a prickling, itching dryness of the throat, hoarseness, later aphonic, with occasionally dysphagia. The cough is distinctly laryngeal unless the repeated hackings induce a spasmodic and expulsive deep cough, but the effort is primarily to free the larynx from the tickling and dry sensation. The laryngeal appearances of the tubercular lesion are fairly characteristic. The color is a shining grayish or yellowish pink, and the location of the lesion is usually in the interarytenoid space, arytenoid bodies, ventricular bands, epiglottis and vocal cords. The interarytenoid space at first shows only a thickening, but later a roughened convexity anteriorly. The arytenoid bodies and ary-epiglottic folds become club-shaped, while the infiltrated ventricular bands often cover the cords, portions of which, especially the processus vocalis, become congested and nibbled by erosions. The turban appearance of the epiglottis follows after other parts are broken down. Diagnosis in the later stages is seldom difficult.

The author is opposed to the routine spraying and inhaling much in vogue, believing in mild treatment and the introduction of solutions which help keep the parts free of mucus. Repeated coughing or hawking should be interdicted as much as possible. The inflammatory condition should receive

such treatment as is indicated, mostly astringents as zinc chlorid, tannate of glycerin, creosote and iodine, pyoktannin and silver nitrate, introduced with the least possible irritation to the parts. The nose and throat should be kept in as good condition as possible. In the strictest sense of the word he does not believe any case is cured, but thinks that with close observation and control of every detail, many patients can live for a number of years. *Richards.*

V.—MISCELLANEOUS.

Development of the Faculty of Speech.

G. HUDSON MAKUEN, Philadelphia. (*International Medical Magazine*, July, 1903.) Ill health is responsible for many cases of faulty development in speech. Among the principle causes are the infectious diseases of childhood, which seem to have a special predilection for the nerve centers of speech and are responsible for many serious disturbances. A severe shock to the nervous system from any cause, a sudden fright or blow upon the head is often an exciting cause of speech defects, especially in children predisposed to such conditions by means of unstable mental and physical constitutions.

To have good speech, a child must be both, well-born and well-bred. He must inherit a tendency toward the development of good speech and must hear only good speech. He must be healthy mentally and physically and free from gaps or weak places in the nervous system. Deformities of structure have considerable influence in speech development; among these are cleft lip or palate, deformities of the alveolar arch and irregular teeth. Hypertrophied tonsils and the various forms of nasal obstruction affect the voice more than they do the development of speech. Many of the causes of disturbance are due to the changes in the central mechanism of speech rather than in the peripheral organs.

Crying is the child's initial form of expression. It appears immediately upon the advent of the child and continues on

a gradually diminishing scale for three years. The initial cry is the cry of an instinctive effort to expand the lungs, develop the vocal organs, and is probably seldom due to pain or discomfort. By the fifth week different kinds of crying sounds have been used, each to denote a special need or state of feeling, and the observant mother soon learns to understand this preliminary child language. Laughing, smiling, and other forms of facial expression begin in the second week; grunting appears in the third.

Most child sounds are at first instinctive and automatic, but soon become imitative. The earliest forms of expression are some of the elementary sounds as *ama*, *mama*. These are the result of unconscious imitation. By the fourteenth month the child begins to echo what it has heard without regard to meaning. This condition is known as *echolalia*.

A little later intelligent speech production begins, a very decided change takes place, and the child may have difficulty in making the transition. It happens occasionally that for a few days all speech is forgotten. This transition is sometimes a slow process and should have careful attention. The conscious mind is endeavoring to take control of the processes of speech, and what was formerly a mere echo of comparatively meaningless words and sentences is now becoming the voluntary oral expression of thoughts, arising in the intellectual centers of the brain. A confusion of thoughts at this time often brings about a lack of harmony in the action of the cerebral speech centers, resulting in the various forms of defects of speech.

Richards.

**Notes from the Throat Department of the Pathological
Laboratory of the Manhattan Eye and Ear Hospital.**

JONATHAN WRIGHT, New York. (*American Journal of the Medical Sciences*, June, 1903.)

- (a) A rapidly recurring bleeding polyp of the septum nasi, appearing twice in a woman, each time at the seventh month of pregnancy.
- (b) Papillary adenomatous hypertrophy of the mucous membrane of the septum.
- (c) A cyst in the lymphoid tissue of the pharynx.

(d) Effusion of serum into the nasal mucosa in coryza.

With remarks on the relation of these lesion to other morbid conditions of the nose and throat.

A pathological study to which the reader is referred, as it does not lend itself well to abstract. One point only may be here referred to, namely: Dr. Wright thinks there are plausible reasons for believing that "the essential lesion and the primary one in atrophic rhinitis is a bone lesion which interferes in some way with the radical vessel, which lie in bony canals, or are compressed in some way against bony plates."

Richards.

Esophagoscopy, and Its Diagnostic Value.

HUGO STARK. (*St. Petersburg Med. Wochen.*, June 28, 1903.) Esophagoscopy is practicable only in the hands of an expert, owing to the severe discomfort which it is likely to occasion the patient. It requires much experience to be able to interpret satisfactorily the pictures obtained. The majority of persons, however, can be examined, and the most suitable instrument is a straight, strong tube. Esophagoscopy is the most certain method for the recognition and localization of foreign bodies. An early diagnosis of carcinoma is only possible with the help of esophagoscope. Anatomical alterations such as ulcers, inflammations, scars, etc., are recognized most certainly by the method. The differential diagnosis between functional and anatomical stenosis is in many cases only possible by the help of the esophagoscope.

Goodale.

Stricture of the Esophagus Following Diphtheria.

JUNGNICKEL. (*Prager Med. Wochen.*, Sept. 17, 1903.) This is an unusual case of stricture of the esophagus. The patient, a man 18 years of age, was taken with diphtheria, and apparently recovered completely in the course of 8 days. Two weeks later difficulty in swallowing began to set in, which showed itself only for solid food. This condition lasted or several months, and was unattended by other symptoms. Four months later, examination showed an obstruction in the

esophagus, eighteen centimeters from the teeth extending for a distance of twelve centimeters. Esophagotomy was performed by Bergman, who found at point corresponding to the obstruction, a cicatricial band, extending obliquely, below and under which a bulging diverticulum was found, evidently due to the repeated passages of the bougie. Examination twelve centimeters lower down showed another stricture, which it was almost impossible to penetrate with the fine urethral sound. Systematic dilatation was performed, and the patient left the hospital eight months later, able to swallow, although still obliged to pass bougies several times a week. The author regards the ulceration and scar formation to be due to the attack of diphtheria. *Goodale.*

Anesthesin in Rhino-Laryngeal Practice.

COUTADE. (*Allg. Wien. Med. Zeit.*, Vol. 48, No. 12.) The author reports results of experiments with this preparation in clinical work. In the case of a young man, whose tonsils were to be cauterized, anesthesin was applied on one side, in the form of powder, with the result that no pain was experienced here, while on the opposite side, the burning caused much discomfort.

In tuberculosis of the larynx, attended with much pain, anesthesin was insufflated, with the result that swallowing was performed without pain for 48 hours, and even eight days later, there was a distinct diminution of sensibility.

Anesthesin is preferably applied in the form of a powder but may be used in painful laryngitis, in the following formula:

Anesthesin	- - - -	20.0
Menthol	- - - -	10-20.0
Ol. Oliv.	- - - -	100.0

Goodale.

Non-Tubercular Hemorrhages of the Air-Passages.

LAFAYETTE PAGE, Indianapolis. (*Jour. Amer. Med. Assn.*, Dec. 13, 1903.) Three cases of severe non-tubercular hemorrhage are reported. In the first there was complete

necrosis of the posterior portion of the septum with ulceration of the veins of the pharyngeal vault. This was of syphilitic origin, the treatment of which condition soon stopped all bleeding. The second case had severe hemorrhage, supposed to come from the stomach, and for which laparotomy was performed and the stomach opened. As the hemorrhage still continued the author was called and found two deep fissures $1\frac{1}{2}$ inches long at the base of the tongue with deep excavation. These were of syphilitic origin and the cause of the hemorrhage, which ceased after anti-syphilitic general and local treatment.

The third case was one of hemorrhage due to angioma on the vocal cord. Here the hemorrhage had been supposed to be due to pulmonary trouble and the patient ordered to Colorado. The angioma was destroyed with the galvano-cautery.

Cocaine spray and suprarenal extract are valuable aids in making a diagnosis.

Richards.

An Analysis of Fifteen Hundred Cases of Tuberculosis Discharged from the Adirondack Cottage Sanitarium from Two to Eighteen Years Ago.

LAWRASON BROWN, Saranac Lake. (*N. Y. Jour. Amer. Med. Assn.*, November 21, 1903.) The first five hundred of these cases were on admission not as favorable as the second thousand, since there were a much greater number of advanced cases. Of the entire number 497 (33 per cent.) are alive; 569 (38 per cent.) are dead; and 434 untraced. Of the 497 patients that are alive, 329 (22 per cent. of the whole) are well; 69 (4.6 per cent. of the whole) are arrested; 44 (3 per cent.) relapsed, and 55 (3.7 per cent.) are chronic.

Richards.

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